INDUSTRIAL MEDICINE *and* SURGERY

Established 1932

JANUARY 1961

Volume 30 Number 1

as powerful as the narcotics in cough suppression... but much longer acting

NON-NARCOTIC

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Chlophedianol HCl

SYRUP

one teaspoonful affords 4 to 8 hours' freedom from cough distress

ULO maintains its maximal cough-suppressant effect undiminished for 4 to 8 hours, thus calling for fewer daytime doses and usually providing freedom from cough distress through the night.

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Write for Physicians' Reference Brochure with full bibliography.

For Children, too

Exceptionally well tolerated; no narcotic overlay; compatible with other indicated medications.

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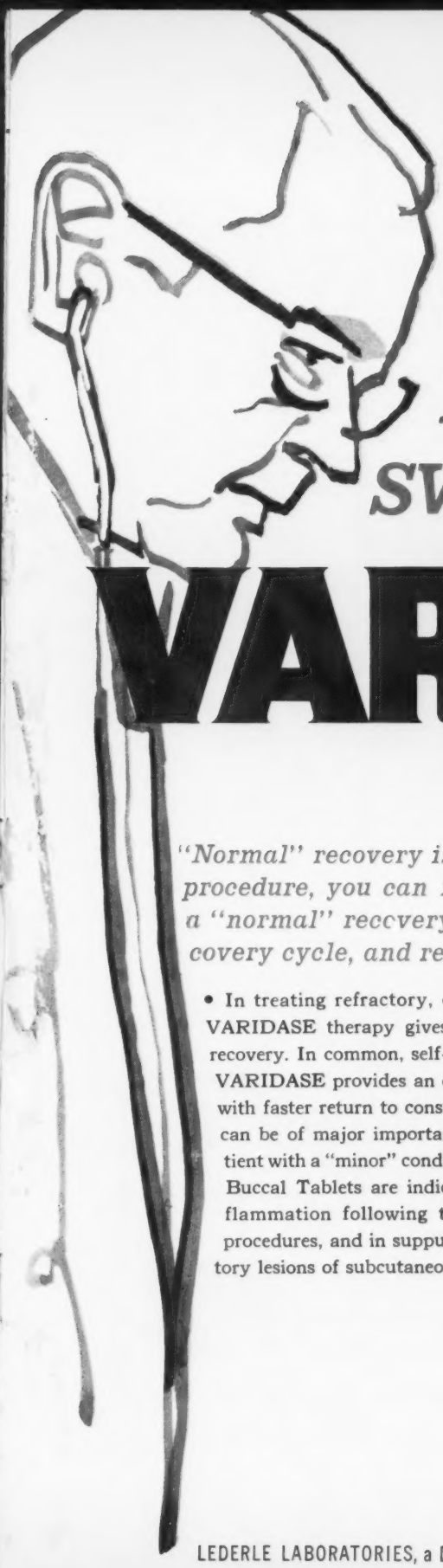
Northridge, California



do all you can whenever there is local inflammation/ swelling/pain...

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buccal tablets

"Normal" recovery is not enough. Now, by adding VARIDASE to your procedure, you can release your patient from the stress and pain of a "normal" recovery—put comfort in convalescence, shorten the recovery cycle, and reap the reward of greater patient appreciation.

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LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

now available in new multi-dose vial

Terramycin®

BRAND OF OXYTETRACYCLINE

INTRAMUSCULAR SOLUTION

conveniently preconstituted for prompt parenteral administration in office or at bedside

new 10 cc. vial permits greater economy, convenience, and flexibility in dosage

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SIDE EFFECTS AND PRECAUTIONS: Aside from occasional mild pain at injection site, adverse reactions (including allergic) have been rare. As with all I.M. preparations, injection should be made within the body of a relatively large muscle. After insertion of needle, aspiration should be attempted before injecting to avoid inadvertent administration into a blood vessel; care should always be taken to avoid injecting into a major nerve or its surrounding sheath. Subcutaneous and fat-layer injection may cause mild pain and induration, which may be relieved by an ice pack.

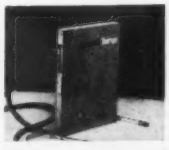
Use of antibiotics may result in an overgrowth of nonsusceptible organisms—particularly monilia and resistant staphylococci. If a new infection caused by a resistant pathogen appears, discontinue the medication and institute appropriate specific therapy as indicated by susceptibility testing.

SUPPLIED: *Terramycin Intramuscular Solution* is available in the new 10 cc. multi-dose vial, providing five 2 cc. doses, 50 mg./cc., and in 2 cc. prescored glass ampules, containing 100 mg. and 250 mg., packages of 5 and 100. For maximum rapidity of effect—*Terramycin Intravenous,* in vials of 250 mg. and 500 mg. (buffered with 1 Gm. and 2 Gm. ascorbic acid, respectively). Available for oral therapy: *Cosa-Terramycin® Capsules,* 250 mg. and 125 mg.; *Cosa-Terrabon® Oral Suspension* (preconstituted), 125 mg. per 5 cc. teaspoonful, in bottles of 2 oz. (60 cc.) and 1 pint; *Cosa-Terrabon® Pediatric Drops* (preconstituted), 5 mg. per drop (100 mg. per cc.), bottle of 10 cc. with calibrated plastic dropper. In addition, a variety of other systemic and local dosage forms are available to meet specific therapeutic requirements.

More detailed professional information available on request.

*Xylocaine® is the trademark of Astra Pharmaceutical Products, Inc. for its brand of lidocaine.

a reservoir of dependable performance— Terramycin® therapy



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INDUSTRIAL MEDICINE and SURGERY

ESTABLISHED 1932

The International Journal of Medicine in Industry

JANUARY, 1961
VOLUME XXX
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BPA

PERSONAL NEWS AND NOTES

W. WAYNE STEWART, M.D., has been named Medical Director of Sun Oil Company. DR. STEWART joined Sun as a consultant in 1952, and has acted as physician in charge of the company's medical program since 1958.

As Medical Director, he will be responsible for carrying out the company's long range preventive medical program, which Sun provides for its 18,000 employees both in domestic and foreign operations.

DR. STEWART is a graduate of Miami University (Ohio) and Hahnemann Medical College, Philadelphia. After serving as a Naval lieutenant commander during World War II, he continued postgraduate work at St. Luke's and Children's Medical Center, Philadelphia, and in 1949 entered private practice as a specialist in chest diseases.

He is on the staffs of Hahnemann and Rush Chest hospitals, Philadelphia, and Crozer and Chester hospitals, Chester, Pa. He is also director of Philadelphia's Penn Center Medical Clinic, which specializes in industrial medicine.

JOHN M. KURKJIAN, M.D., resident physician in surgery and industrial medicine at Massachusetts Memorial Hospital, has beer appointed Manager of Health Services for Raytheon Company's Industrial Components Division, Newton, Mass. He received his B.S. degree from Tufts University, an M.S. from the University of Illinois, and his M.D. from Boston University. He served at Massachusetts Memorial Hospital for two years before coming to Raytheon.

American College of Allergists will hold its Graduate Instructional Course and Seventeenth Annual Congress, March 12-17, 1961, the Statler Hilton Hotel, Dallas, Texas. For information write: JOHN D. GILLASPIE, M.D., Treasurer, 2141 14th Street, Boulder, Colorado.

Industrial Medicine and Surgery has been awarded a Certificate of Merit, for the June, 1960, feature issue on the McIntyre Research Foundation Conference on Silicosis, in the 22nd annual editorial achievement competition sponsored by *Industrial Marketing*. Of 703 entries in the 1960 competition, there were 46 winners.

Industrial Medicine and Surgery's Certificate was one of three awards in the best single issue group of the Class, Institutional, and Professional Publications classification.

CHARLES E. DUTCHESS, M.D., Consultant Editor, accepted the Certificate of Merit for *Industrial Medicine and Surgery* at a luncheon in New York City on November 29.

CHARLES E. DUTCHESS, M.D. (right) Consultant Editor of Industrial Medicine and Surgery, accepted a Certificate of Merit Award from Mr. S. R. BERNSTEIN, Editorial Director of Industrial Marketing, at a luncheon honoring winners of Industrial Marketing's 1960 editorial awards at the Waldorf-Astoria Hotel in New York City on November 29.

The American Academy of Occupational Medicine will hold its annual meeting February 8-10, 1961, at the Statler Hotel in Detroit. J. A. QUIGLEY, M.D., President of AAOM, will preside at the meeting.

The Gehrman Memorial Lecture will be given by THEODORE C. WATERS, LL.D., Baltimore, Md., on the subject "Trends in Compensation Legislation and Administration." The banquet speaker will be KENNETH MCFARLAND, Ph.D., Educational Consultant to the General Motors Corporation at Topeka, Kansas.

Scientific papers scheduled for the meeting include:

"Fundamental Principles of Biostatistics" by FELIX E. MOORE, M.D., University of Michigan, Ann Arbor; "Fundamental Principles of Epidemiology" by ALEXANDER G. GILLIAM, M.D., National Institutes of Health, Bethesda, Maryland; "Epidemiological Methods and Community Air Pollution" by JOHN J. PHAIR, M.D., University of Cincinnati; "Application of Biostatistics and Epidemiology to Industrial Population" by THOMAS MANCUSO, M.D., Ohio State Department of Health, Columbus.

"What Pulmonary Function Testing Can Tell the Industrial Physician" by BENJAMIN W. LEWIS, M.D., Wayne State University, Detroit; "Application of Functional Tests to Pulmonary Disease Entities of Occupational Origin" by DAVID V. BATES, M.D., McGill University, Montreal.

"Occupation and Pulmonary Emphysema" by GEORGE W. WRIGHT, M.D., St. Luke's Hospital, Cleveland; "Occupation and Cardiac Impairment" by RICHARD J. BING, M.D., Wayne State University, Detroit; "Auditory Problems in Occupational Medicine" by MERLE LAWRENCE, Ph.D., University of Michigan, Ann Arbor.

"Skin Cancer" by LEO J. MIEDLER, M.D., University of Michigan, Ann Arbor; "Evidence for Radiation and Chemicals as Leukemo-

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For neuralgias, dysmenorrhea, upper respiratory distress, and postsurgical conditions—new compound of Soma, phenacetin and caffeine kills pain, stops tension, reduces fever—gives more complete relief than other analgesics...acts fast, relief lasts four to six hours.

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genic Agents" by EUGENE P. CRONKITE, M.D., Brookhaven National Laboratories, Upton, Long Island; "Lung Cancer" by ARTHUR J. VORWALD, M.D., Wayne State University, Detroit; and "Trauma and Cancer" by ALAN R. MORITZ, M.D., Western Reserve University, Cleveland.

WILLIAM P. SHEPARD, M.D., who is retiring shortly as Chief Medical Director of the Metropolitan Life Insurance Company, has accepted the office of Associate Director of the Institute for Advancement of Medical Communication, a non-profit organization engaged in developing improved methods for intra- and interprofessional communication in the health sciences. His appointment was announced by RICHARD H. ORR, M.D., Executive Director of the Institute.

DR. SHEPARD has long been active in the fields of preventive medicine and occupational health and is at present Chairman of the Council on Occupational Health of the American Medical Association.

EDWARD V. HENSON, M.D., has been appointed to the new position of medical research associate in the general office medical department of Standard Oil Company (Indiana).

He was formerly Medical Director of the Union Carbide Chemicals Company plant at South Charleston, West Virginia. He joined that company as staff physician in 1947, following service as a captain with the U. S. Army Medical Corps.

DR. HENSON is assistant editor of the *Journal of Occupational Medicine*, a Fellow of the Industrial Medical Association, and a member of the American Medical Association, American Public Health Association, and American Industrial Hygiene Association. He was graduated with a bachelor of science degree from Pennsylvania State College, and received his medical degree from Jefferson Medical College.

JOHN M. BISHOP, M.D., has been appointed Assistant Medical Director of the Manhattan-Bronx-Westchester Territory of the New York Telephone Company.

DR. BISHOP received his medical degree from the University of Illinois in 1948. Following graduation from medical school he joined the United States Public Health Service and interned at the Public Health Service Hospital, Seattle, Washington.

In 1953, DR. BISHOP was assigned to Europe with the PHS as the physician for the American Embassy, Paris, France. In 1954, he was appointed Medical Officer in Charge of the PHS medical activities under the Refugee Relief Program in Europe, North Africa and the Middle East.

Late in 1956, he returned to the U. S. to become Deputy Director of the Public Health Service Hospital in San Francisco. The following year he resigned from the Service to enter private practice as a surgeon. At the time of his discharge from active duty he held the rank of Senior Surgeon (Commander).

He practiced in Seattle for two years and during this time also worked as a medical hospital consultant in the Pacific Northwest designing and planning community hospitals. In 1959, he moved to New York to work as the Deputy Secretary General of the World Medical Association. Later, he served as an international medical program consultant for the American Medical Association and other related medical groups. He was the author of a 300 page survey report prepared for the AMA on international medical and health activities of American organizations.

ALFREDO SALAZAR has been appointed to the new position of Industrial Hygiene Chemist in the office of the Medical Director of Standard Oil Company (Indiana). He will develop and operate a chemical-engineering laboratory designed to support industrial hygiene activities throughout the company.

MR. SALAZAR was formerly Industrial Hygiene Engineer for the Navy Department at the San Diego, Calif., air station and Boston, Mass., shipyard, and Associate Industrial Hygienist for the State Health Department of West Virginia.

He received a chemical engineering degree from the National School of Engineering in Lima, Peru, and attended Harvard University under a Rockefeller Foundation fellowship. He holds the rank of lieutenant commander as a reserve officer of the United States Public Health Service.

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Supplied: Scored, pink tablets, bottles of 50. Each tablet contains PARAFLEX® Chlorzoxazone† 125 mg., and TYLENOL® Acetaminophen 300 mg.

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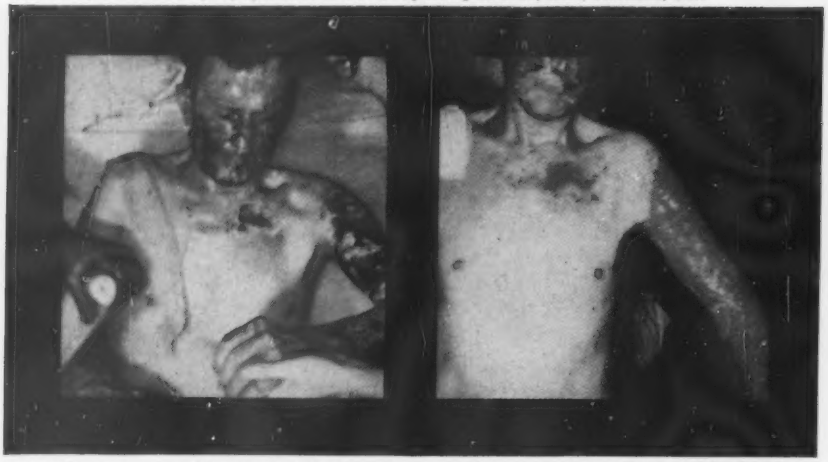
From the film—"Skin Grafting of Extensive Burns" by Harry R. Grau, M.D., Cleveland, Ohio



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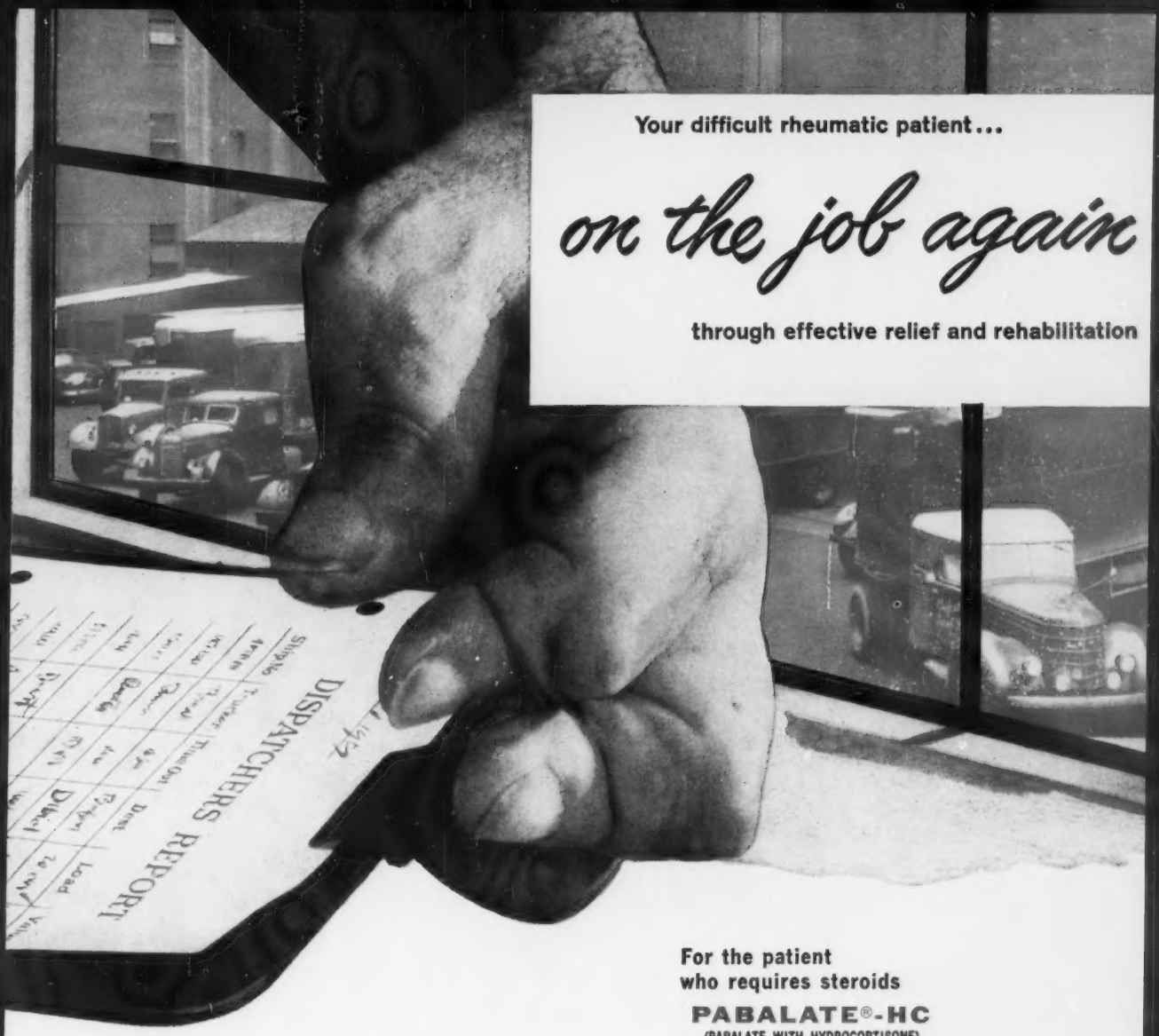

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For the patient who does not require steroids

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Potassium para-aminobenzoate.. 0.3 Gm.
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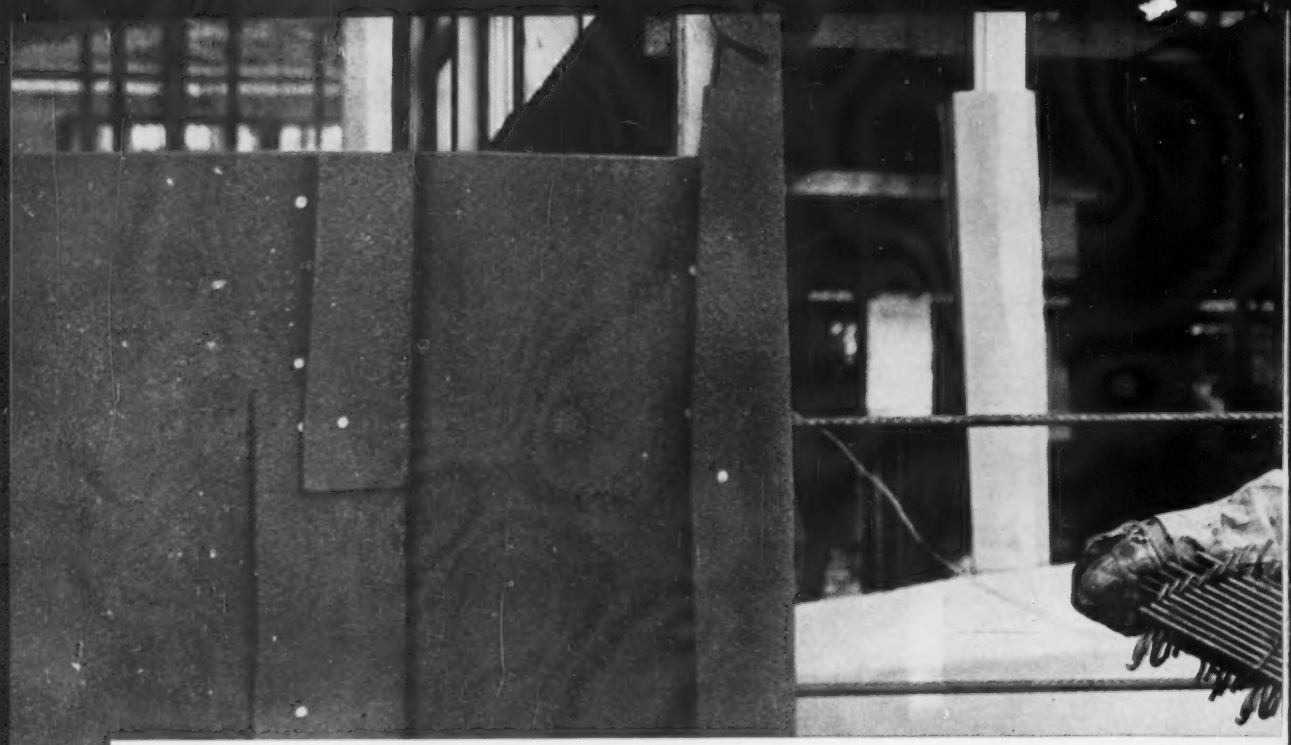

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References: Annals of Allergy, Vol. 6. July-August 1948. Study of a New Histamine Antagonist, The Pennsylvania Med. J., Vol. 51. Clinical Study of a New Antihistaminic Drug—Phenindamine. Dristan Clinical Study.

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in cough control, too—
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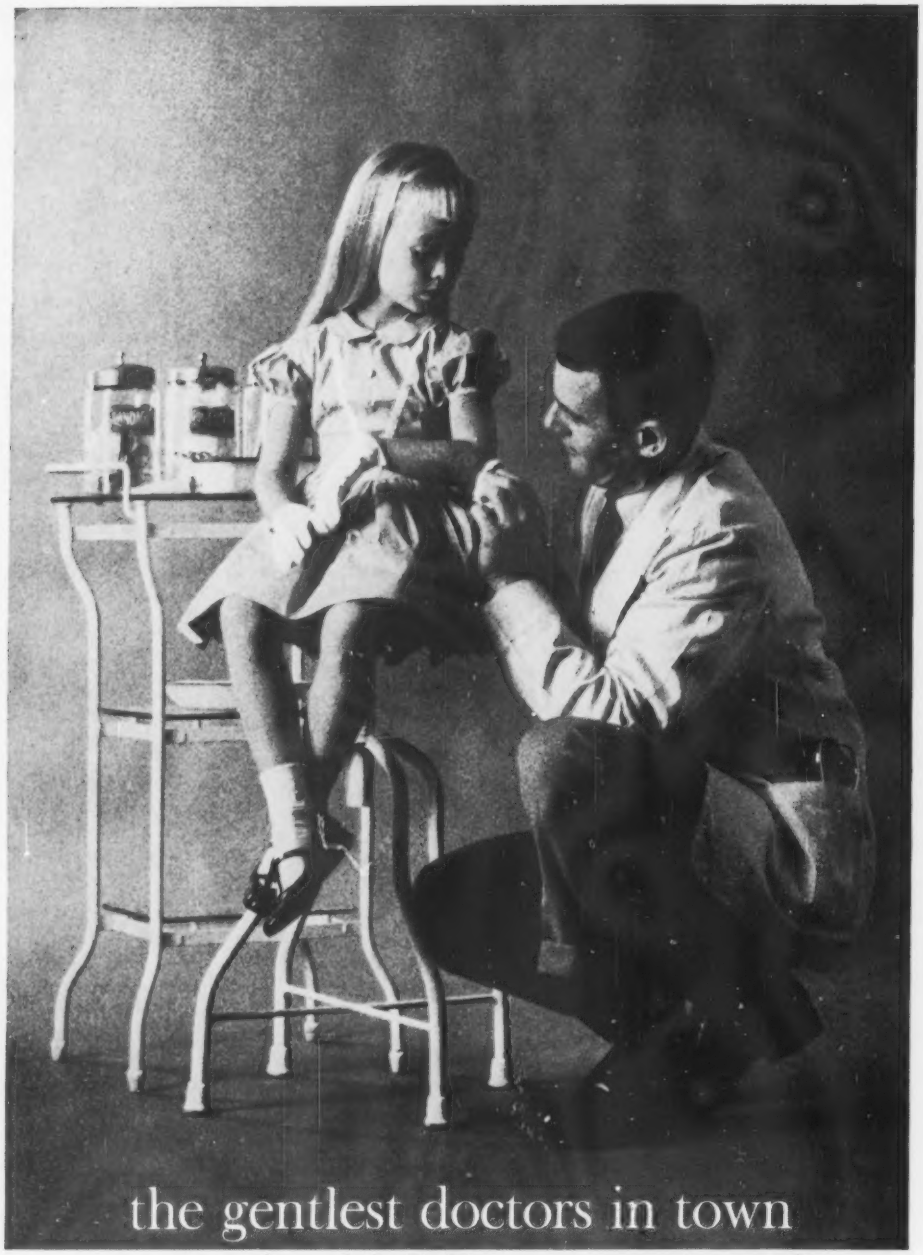

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References: 1. DeNyse, D. L.: *M. Times* 87:1512, Nov., 1959. 2. Ganz, S. E.: *J. Indiana M. A.* 52:1134, July, 1959. 3. Gruenberg, Friedrich: *Current Therap. Res.* 2:1, Jan., 1960. 4. Kearney, R. D.: *Current Therap. Res.* 2:127, April, 1960. 5. Lichtman, A. L.: *Kentucky Acad. Gen. Pract. J.* 4:28, Oct., 1958. 6. Mullin, W. G., and Epifano, Leonard: *Am. Pract. & Digest Treat.* 10:1743, Oct., 1959. 7. Shanaphy, J. F.: *Current Therap. Res.* 1:59, Oct., 1959. 8. Collective Study, Department of Medical Research, Winthrop Laboratories. 9. Hergesheimer, L. H.: An evaluation of a muscle relaxant (Trancopal) alone and with aspirin (Trancoprin) in an industrial medical practice, to be submitted.

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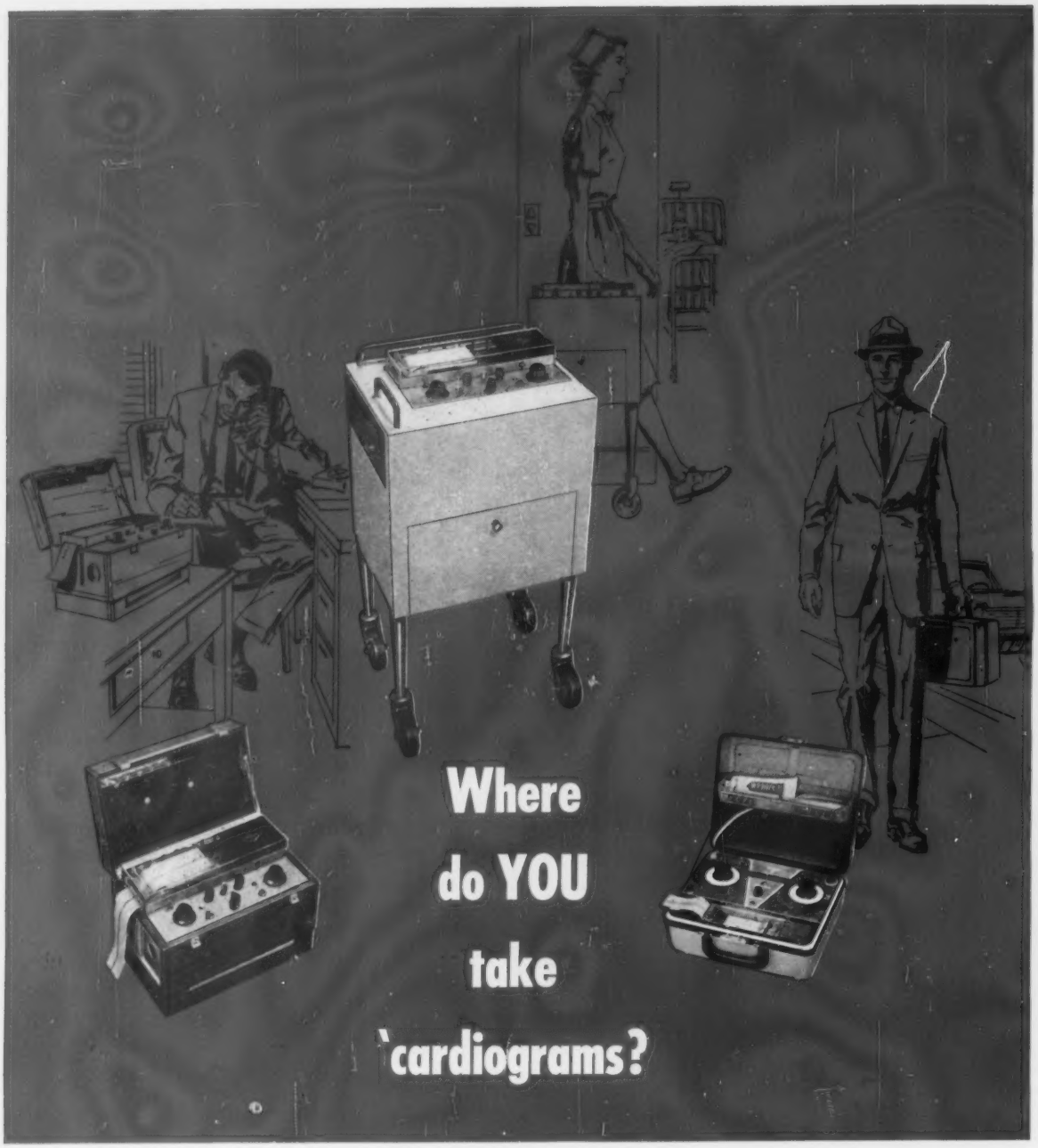

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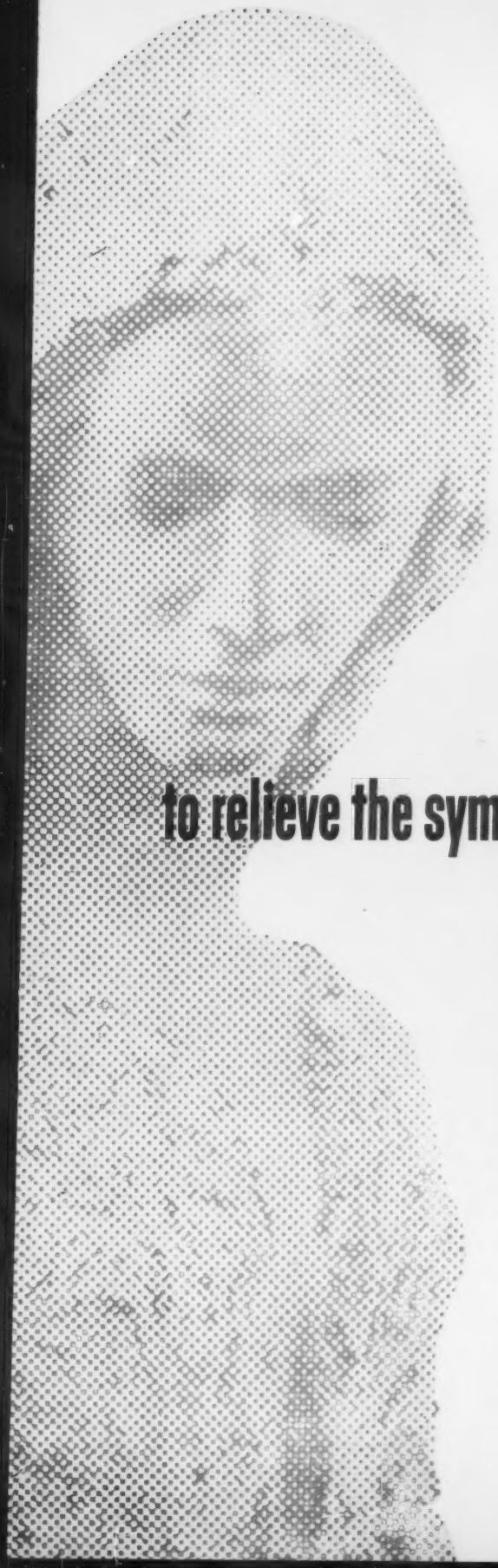

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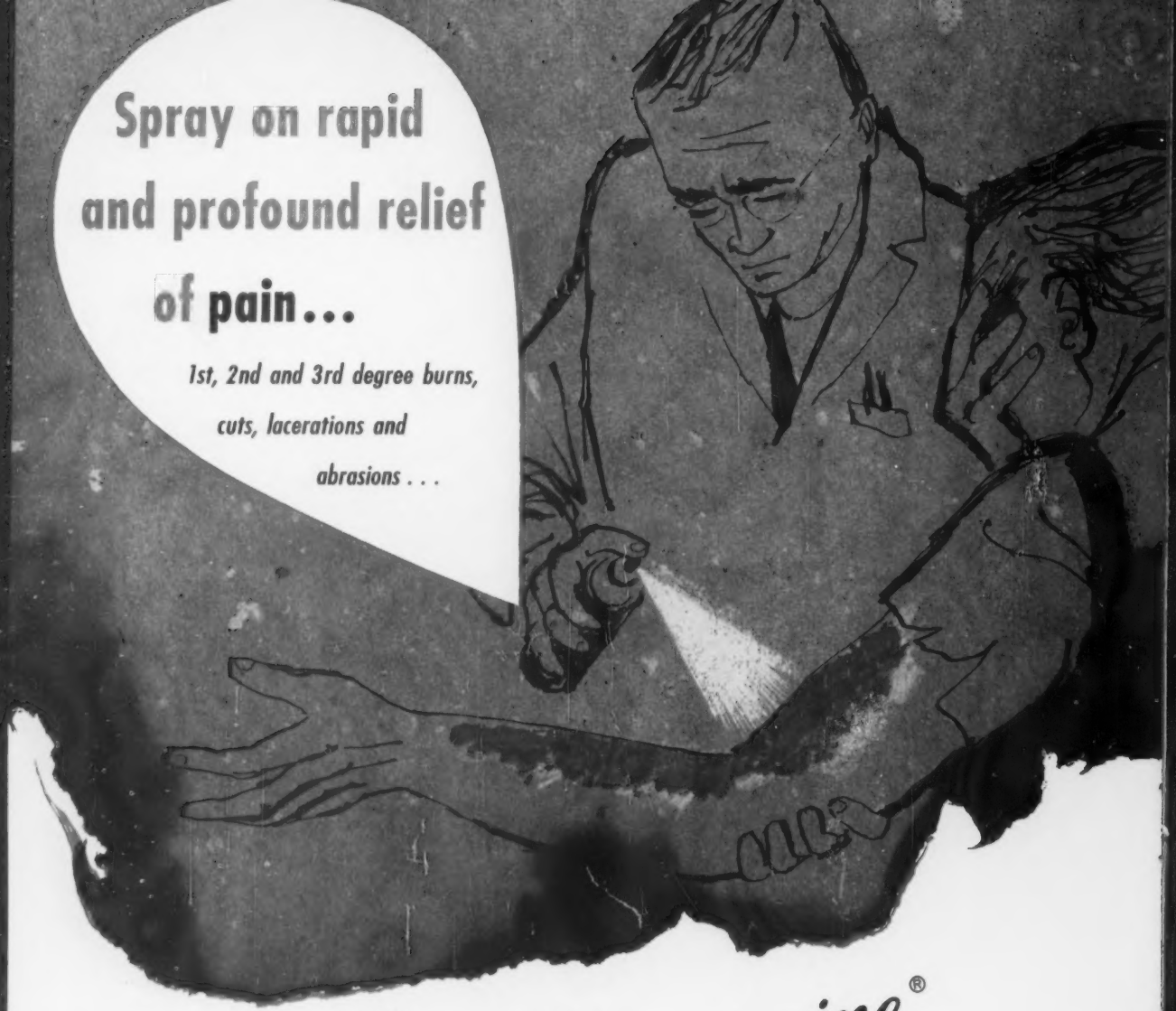

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1. Kuba, E. F.: First Stage Treatment of Burns and Prevention of Shock, Indust. Med. 12:369-371 (Aug.) 1958.

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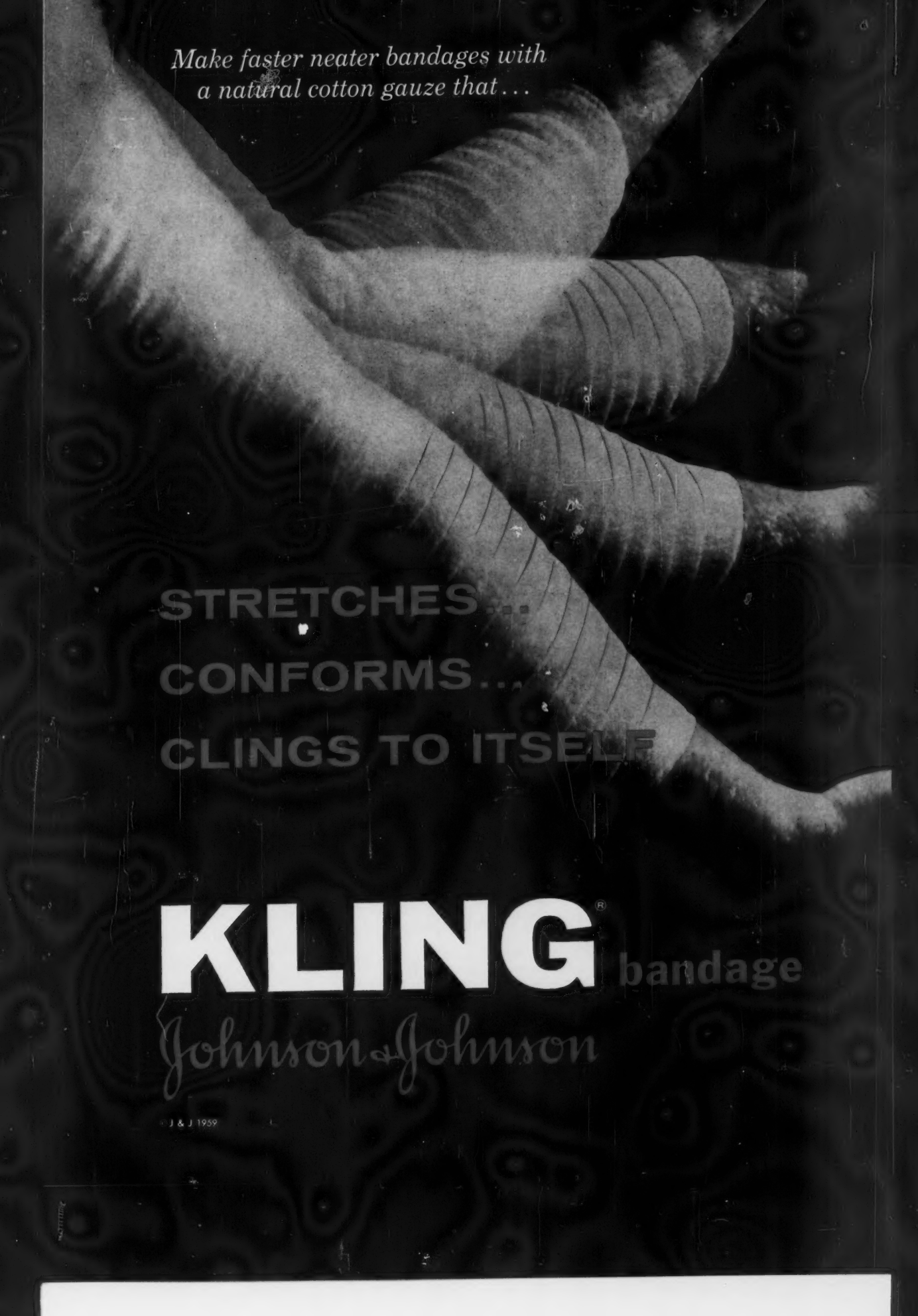
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UNUSUAL CAUSES OF NERVE INJURIES

• A. A. MARINACCI, M.D.
Los Angeles, California

Telephone cradlers, television watchers, carpet layers and hodcarriers may suffer from unusual pressure neuropathies that are often misdiagnosed.

The key to diagnosis of the neuropathies is twofold: the reenactment of the situation by which the paralytic state was produced, and the use of the electromyogram to determine the precise location of the lesion.

Pressure neuropathies of themselves constitute one of the large group of peripheral nerve disorders. Most are fairly common and the most typical ones are usually readily recognized by the clinician. But even some of the common ones can cause a considerable amount of diagnostic difficulty. This difficulty arises from the fact that the average physician does not see a sufficient number of these cases to become familiar with them. When a given case is encountered, he is often perplexed as to the cause of the paralysis. This perplexity arises from the fact that the patient is greatly upset and describes a complex picture arising from many suggestions from the family, neighbors, and, above all, by other physicians who have not recognized the syndrome. It is not uncommon for patients affected with a peripheral neuropathy to be considered as a case of brain tumor, stroke, or other central nervous disease including multiple sclerosis. This clinical complexity is more acute when there is more than one pressure neuropathy present, especially when it involves both the upper and lower extremities of the same side. On the other hand if the lesion is on the opposite upper and lower extremities, it may suggest multiple sclerosis or spinal cord tumor. The diagnosis becomes even more difficult when a pressure neuropathy does not occur in a common anatomical position. The patient is submitted to other diagnostic procedures, in most instances with negative results. Finally, when the electromyography survey is accom-

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plished, the clinical picture of the paralysis is usually clarified.

In the following paragraphs will be discussed a number of specific, though at times unusual, pressure neuropathies with case presentations: (1) unusual instances of progressive Bell's Palsy, (2) cervical plexus injuries in hod carriers, (3) axillary nerve, upper trunk of the brachial plexus and C5 nerve root compression, (4) mechanics of crutch paralysis, (5) musculocutaneous nerve paralysis from carrying heavy bundles, (6) triad paralysis from "dependre arms," (7) paralysis of the inveterate television watcher, (8) hammer-hand syndrome, (9) sciatic stretching in carpet layers, (10) sciatic-femoral nerve combinations, and (11) plantar nerve injury.

Progressive Bell's Palsy

A degree of paralysis of the seventh cranial nerve is rather common. The anatomical position of this nerve is vulnerable when passing through the bony facial canal. It is at this point (stylomastoid foramen) that the lesions of this nerve are most common. The electromyogram in cases of Bell's Palsy usually reveals characteristic anatomical changes. The denervation activity is distributed to all the muscles (frontalis, orbicularis oculi, orbicularis oris, and the auricular muscles group). In short, the denervation activity in Bell's palsy is recorded in all muscles supplied by this nerve, for the lesion takes place in the facial canal before the division of the branches takes place.

The unusual causes present an entirely different clinical picture, for here one may have an involvement of one single branch, resulting in weakness of a restricted muscle group such as the frontalis, orbicularis oculi or only the muscles of one side of the mouth (orbicularis oris). These isolated pressure neuropathies of the seventh cranial nerve can simulate a myasthenia gravis when only the orbicularis oculi is involved, giving arise to ptosis of the eyelid. When the cervical branch innervating the orbicularis oris alone is involved, this may simulate an intracranial lesion.

These isolated pressure neuropathies of the seventh cranial nerve have been found most common in people who place the receiver of the telephone against their ear causing the receiver to press against either the cervical or temporal branch of the seventh cranial nerve. This usually occurs in patients whose work requires the use of the telephone for long periods, and while doing so, rest the base of the earphone on the shoulder while the rim of the earphone rests against the side of the face leaving the hands free for writing messages, as demonstrated by the following case:

Case 1. A 32 year old male began to complain of ptosis of the left eye. This weakness led the first consultant to believe that the patient had a myasthenia gravis. However, four or five weeks later, such weakness progressed to the left side of the mouth. When the second neurological consultant saw the patient because of the progression of the weakness, it was thought to be the result of an intracranial lesion. An EEG was normal and the patient was referred to a neurosurgeon for an angiogram. An electromyogram was requested which revealed partial denervation of the left orbicularis oculi and the left frontalis and the orbicularis oris. The auricular group of muscles were normal denoting that the lesion had taken place distal to the facial canal.

Comment: The EMG findings suggested that the lesion was outside the cervical canal and led to an inquiry into the patient's occupational habits. He said that he worked as a clerk in a meat warehouse, where he constantly took orders over the telephone, placing the receiver on the left shoulder. The rim of the receiver, resting against the side of the face, caused pressure first on the temporal and then on the cervical branches of the seventh cranial nerve. The patient was told to change his work habits and to use a head piece telephone receiver. The patient was seen at monthly intervals for six months, and gradually recovered from his facial weakness.

Cervical Plexus Injury in Hod Carriers

Injury to the cervical plexus is not uncommon. Among the several causes is the bearing of heavy weights on the shoulders resulting in stretching of the cervical plexus as well as the 11th spinal accessory nerve. However, injury to the cervical plexus in hod carriers, (plasterers, brick-layers, etc.) presents an unusual pressure neuropathy. The onset is usually gradual and the patient is frequently diagnosed a psychoneurotic, for the complaint cannot be justified on the basis of the physical findings. The patients usually complain of tiredness and muscle spasms in the region of the trapezius. Some of them are suspected of having herniation of either C3 or C4 disk. When atrophy finally appears without associated sensory changes, the patient may be diagnosed as having muscular dystrophy. The following case is illustrative:

Case 2. This 37 year old hod carrier began to complain of muscular weakness in the muscles of the left shoulder girdle. At the beginning, it was not apparent that atrophy was taking place and no definite muscular weakness could be demonstrated. The patient continued to complain, and ultimately atrophy of the shoulder girdle was seen on the left side. The clinician suspected

either a C3, C4 or C5 nerve root lesion. Opaque myelography was normal. Eventually, an electromyogram revealed denervation activity restricted in areas of the left trapezius. The paraspinal muscles were normal as was the supra-infraspinatus group. Thus, the EMG was consistent with an isolated lesion of the cervical plexus.

Comment: With the aid of the electromyogram the lesion in the cervical plexus was identified. Further, it indicated that the atrophy of the left supra-infraspinatus was that of disuse, as only normal activity was found electromyographically. On further investigation, it was discovered that the patient's occupation was that of hod carrier, an occupation in which considerable pressure is exerted on the shoulder. It would have been difficult, on the basis of the clinical manifestations, to distinguish between atrophy of disuse and neurogenic atrophy due to the cervical plexus lesion. The big fault with the clinical study in this case was failure to learn the patient's occupation which should have suggested his symptoms.

Pressure Neuropathies of the Axillary Nerve, Upper Trunk of the Brachial Plexus and C5 Nerve Root Compression

An isolated lesion of the axillary nerve is rather common. It usually results from a blow directed at the posterior aspect of the arm at the level of the surgical neck of the humerus, or more commonly as a result of posterior dislocation of the shoulder joint. The electromyograms in these cases show denervation activity confined to the deltoid and teres minor. The rest of the nerves are normal. However, a similar type of deltoid paralysis is encountered in involvement of the upper trunk (Erb's paralysis) of the brachial plexus. Clinically, the manifestations of these two lesions are difficult to differentiate. However, in Erb's paralysis, denervation is found in the supra-infraspinatus, deltoid, biceps and brachioradialis. No changes are found in the paraspinal muscles. The distribution of this denervation therefore distinguishes a pure axillary nerve lesion from an Erb's paralysis. Moreover, severe involvement of C5 nerve root gives rise to paralysis of the deltoid. In many of these cases, the patient is diagnosed as having an isolated axillary nerve lesion. One can readily appreciate the problem represented by these three specific lesions and the fact that, clinically, it is difficult to differentiate them one from the other. In C5 nerve root involvement, denervation is present in the paraspinal muscles of C4-5 level, in the supra-infraspinatus, deltoid and the brachioradialis, with little in the biceps. This problem is demonstrated by the following case:

Case 3. This 33 year old male presented atrophy of the left deltoid muscle with some pain radiating into the corresponding upper extremity. It was thought that some atrophy of the supra-infraspinatus muscles existed. Compression of the C5 nerve root was suspected. Another consultant thought the patient had an Erb's paralysis because of an uncertain history of injury. Opaque myelography revealed no abnormality. An electromyogram, as a last resort, revealed an isolated axillary nerve lesion. Atrophy of the left shoulder girdle was that of disuse.

Comment: With the aid of the electromyogram, the specific lesion was demonstrated and the other diagnosis such as Erb's paralysis and C5 nerve root compression were ruled out. Again, atrophy of the shoulder girdle incident to disuse had misled the clinician to believe there was a nerve root lesion, or an involvement of the upper trunk of the brachial plexus.

Mechanics of Crutch Paralysis

The mechanics of crutch paralysis are of great interest because these patients suffer from partial wrist drop and weakness of the forearm and hand, presenting a picture of either cervical cord tumor, cervical nerve root compression, syringomyelia, or even that of progressive spinal muscular atrophy. It has been the experience of the writer, however, that crutch paralysis produces rather specific neuropathy as demonstrated below:

Case 4. This 22 year old female was obliged to use crutches because of a fractured ankle. She began to complain of tingling sensations and numbness in the forearms and hands. The industrial surgeon thought her to be hysterical. However, another neurologist believed that she might have, in addition to the broken ankle, a cervical cord condition. An electromyogram showed that the denervation activity was confined in the triceps, extensors, flexors, thenar and hypothenar groups on the right and left. The deltoid and biceps were normal bilaterally. Therefore, the findings were those of involvement of the radial, median and ulnar nerves bilaterally at the level of the axilla.

Comment: The writer has seen many patients with a crutch paralysis presenting about the same clinical picture in which the axillary and musculocutaneous nerves are spared, while the radial, median and ulnar nerves in the arm, forearm and hand are involved. With the aid of the electromyogram, one can determine a definite level at which the three nerves are involved.

Musculocutaneous Nerve Paralysis from Carrying Heavy Bundles

An isolated lesion affecting the musculocutaneous nerve is not common. Therefore, when it does occur, it frequently misleads the diagnostician

into believing that he is dealing with a lesion in the cervical region. This stems from the fact that, in addition to the weakness of the biceps, there also is numbness in the forearms and thumb in the cutaneous distribution of the musculocutaneous, suggesting a lesion of the cervical region.

Case 5. A 27 year old housewife returned from the market carrying her bundle in the right arm and leading her child with the left. On climbing the stairs, she suddenly dropped her packages. Following this episode, she first noticed numbness and tingling in the fingers of the right hand. This was interpreted by her physician as herniated cervical disk, and she was admitted to the hospital for cervical traction. Opaque myelography was contemplated. However, the patient's husband objected to this procedure, and requested an electromyogram. This study, four weeks after the onset of the condition, revealed an isolated involvement in the right biceps muscle. The rest of the nerves were normal as were all the cervical nerve roots. On monthly rechecks, definite regeneration was demonstrated, with eventual full recovery as far as the biceps muscles were concerned. However, some defect of sensation in the right thumb was still demonstrable eight months later.

Comment: This case demonstrates how the electromyogram is able to differentiate between a cervical lesion and a peripheral nerve lesion, which is at times difficult to do from the clinical findings alone.

Triad Paralysis from "Dependre Arms"

The writer has seen 11 patients suffering from simultaneous compression of the ulnar, median and radial nerves on the medial aspect of the arm while the patient was under the influence of drugs or alcohol and allowed his arm to hang over the side of the bed or chair, thereby exerting pressure on these nerves. Unfortunately, the lesion can occur at different levels. If at the axilla, it will involve all three nerves. In the middle of the arm, the median nerve is affected more than the radial and ulnar nerves because the radial nerve has already begun its detour around the radial groove of the humerus. If the level of the pressure has been above the antecubital fossa, again the medial nerve will be most affected, because the ulnar nerve has already begun to follow its anatomical course around the olecranon. These patients present difficult diagnostic problems as demonstrated by the following case:

Case 6. This 58 year old man, on recovering from an alcoholic spree, became aware of a severe pain in the right forearm and hand. His physician thought that the patient had a lesion in the cervical region. Three weeks later, another consultant saw definite atrophy of the right shoulder girdle as well as of the right forearm and hand. The patient continued to have severe pain in the hand (causalgia) and required a considerable amount of sedation for relief of this pain. Cervical exploration was being contemplated for cord tumor or nerve root compression. The electromyogram revealed denervation activity confined in the flexors and all the extensors and muscles of the hand on the right. The distal half of the triceps also was involved. The muscles of the neck and shoulder girdles were normal. The EMG indicated a definite lesion existing in the right radial, median and ulnar nerves at the level of the middle of the medial aspect of the arm.

Comment: Here again, atrophy of disuse of the shoulder girdle was misleading to the clinician. The electromyogram was able to establish the presence of a pressure neuritis of all three nerves. The patient continued to have pain although gradual improvement took place. Eighteen months later, he had only slight residuals. When examined two years later, full recovery had taken place.

Paralysis in the Inveterate Television Viewer

Since the advent of television, certain types of multiple pressure neuropathies have become prominent. Perhaps the most common of these attributed to this pastime is bilateral ulnar nerve involvement, incident to resting the elbows on the arms of a chair for prolonged periods. However, involvement of the tibial nerves secondary to hanging the leg over the edge of a reclining chair producing pressure on these nerves at the popliteal fossa also may be seen.[1] The unusual clinical picture represented by one of these pressure neuropathies is indicated in the following case:

Case 7. A 63 year old male developed a progressive atrophy of the muscles of the hands. It was therefore believed that the patient was afflicted with a progressive spinal muscular atrophy. However, an electromyogram revealed an isolated ulnar nerve involvement bilaterally at the level of the elbows.

Comment: The electromyogram was able to differentiate between a myotrophic lateral sclerosis and an isolated peripheral neuropathy by demonstrating the denervation to be essential peripheral. Had the patient had a spinal muscular atrophy, the denervation would have been widespread in all the muscles of the extremities as well as those of the trunk.

Hammer Hand Syndrome

One of the most misleading and unusual lesions

in the group of pressure neuropathies result from using the hand as a hammer, a condition not uncommon among automobile mechanics. This habit may produce two lesions: ulnar nerve involvement at the palm of the hand, and median nerve involvement at the base of the thumb. Of the two, the ulnar nerve lesion in the palm represents the most difficult diagnostic problem. As the patient uses his hand to hammer, he traumatizes those branches of the ulnar nerve that innervate the first dorsal interosseous, producing atrophy of this muscle without sensory changes. These patients have been considered to have progressive spinal muscular atrophy, cervical cord pathology, and even muscular dystrophy, because of the intact sensation.

Case 8. A 24 year old mechanic presented himself with atrophy of the first dorsal interosseous on the right. A distal form of muscular dystrophy was considered as the cause or even a cervical nerve root compression. An electromyogram revealed denervation confined only to the first, second and third dorsal interossei on the right. The muscles of the cervical region, shoulder girdle, arm, forearm and median distribution in the hand were entirely normal. Thus, the EMG findings were consistent with an isolated pressure neuritis, involving the terminal branches of the right ulnar nerve in the palm of the hand.

Comment: In order to explain this isolated lesion, the patient was questioned further and it was discovered that habitually he used his right hand as a hammer. Thus, with the aid of the electromyogram, both the myopathies and a progressive neuropathy were eliminated as the cause of the patient's symptoms.

Sciatic Stretching in Carpet Layers

A special form of pressure neuropathies occurs in patients whose occupation is that of carpet layer. By standing mostly on their toes in the course of their work and using the hamstrings as a spring, they produce stretching of the sciatic nerve bilaterally.

Case 9. This 23 year old male complained of numbness in the feet and radiating pain in the calves of the legs. An involvement of the S1 nerve root was suspected. However, opaque myelography was normal. An electromyogram revealed the paraspinal muscles, the gluteal group, the quadriceps and abductors to be essentially normal. However, moderate changes were present in the hamstrings and muscles of the personal and tibial components of the sciatic nerve, including the intrinsic muscles of both feet. The diagnosis was that of bilateral sciatic nerve involvement.

Comment: This condition is easily diagnosed electromyographically. However, clinically, it presents a difficult diagnostic problem. If the patient has suffered herniation of the fifth lumbar intervertebral disk, the denervation activity would have been present in the entire myotome rather than confined in the distribution of the sciatic nerve alone.

A Combined Sciatic-Femoral Neuropathy

The combined stretching of both the sciatic and femoral nerves is not common, but when it occurs, it misleads the physician to believe that its cause is in the pelvis or cauda equina, because of weakness of the quadriceps and the muscles of the thigh and leg. As the patient lies unconscious as a result of either drugs or alcohol, he may hang the leg over the side of the bed, producing simultaneously pressure on the sciatic nerve and stretching of the femoral nerve.

Case 10. On awaking after an alcoholic bout, this patient noted paralysis of the right lower extremity, and it was thought he had suffered a stroke. Another consultant considered the lesion to be in the cauda equina. Still another thought the patient had a lumbosacral plexitis. Electromyographically the paraspinal muscles and the pelvic girdle were normal; a large amount of denervation was found in the quadriceps, hamstrings, and the muscles of the right leg and foot indicating involvement of the femoral and sciatic nerves. The level of the involvement was the gluteal fold.

Comment: On further questioning, the patient finally revealed that he had become intoxicated and that the paralysis was first noted on awaking from sleep.

Plantar Nerve Paralysis from Brake Injury

Pressure neuritis of the plantar nerve is common. As explained previously,[2] one of the most unusual causes is the result of driving a car without shoes. When a patient was involved in a collision under these circumstances, she spontaneously exerted severe pressure on the plantar nerve while striking the brake with her foot.

Case 11. A 33 year old female complained of a low back pain radiating back of the leg down to the right foot. These symptoms were of six months' duration. Occasionally, the radiating pain down to the left foot was severe in degree. The attending physician believed that the patient had a herniated L5 disk, pressing on the right S1 nerve root. An opaque myelography revealed no diagnostic abnormality. The electromyograms of muscles supplied by the anterior and posterior primary divisions of the first lumbar through the third sacral nerve roots were examined bilaterally. No denervation was recorded in the muscles of the back and the

lower extremities with the exception that 50% denervation was recorded in the intrinsic muscles of the right foot supplied by the medial and the lateral plantar nerves. The EMG pointed to a lesion of the right plantar nerves at the navicular level. A surgical exploration revealed that the plantar nerve at the navicular level was somewhat hardened and bound down by scar tissue. The nerve was freed from the scar tissue and neurolysis was done. The patient was relieved of the radiating pain, as well as the pain in the right foot.

Comment: Investigation revealed that the patient's symptoms started with a painful foot following an automobile accident when she was driving without her shoes on. She was involved in a collision and jammed her foot forcefully against the brakes. Because of the painful foot, other muscle groups (lumbosacral, gluteal, hamstrings and those of the calf) were called to compensate for the lesion of the nerve of the foot, producing compensatory symptoms distracting the attention of the clinician from the true site of the trouble. The diagnosis in this case would have been a simple one if the reenactment of the accident was brought to light.

Comment

It is apparent that the key to the diagnosis of the neuropathies is the reenactment of the cause of the neuromuscular disability. How did the accident that caused the disability arise? This concept of determining the mechanism of injury has been pointed out by Nielsen (1959).

An acute appearance of paralysis incident to a mononeuropathy is usually the result of sudden pressure or stretching, or a direct blow to the region. On the other hand, neuropathies that have a gradual onset are usually due to some prolonged abnormal posture.

In the more chronic development of mononeuropathies, it is important to investigate the occupation of the individual, even to asking about routine postures or the use of the extremities, particularly the one in question. The habitual use of the legs in sedentary workers often gives the clue to the etiology of the paralysis. The acute onset of painful foot often may be accounted for by an injury prior to the onset of the symptoms. Sometimes the patient may report an excessive application of pressure to the plantar surface of the foot involved.

It is pertinent to suggest that the clinician refrain from the early use of the more technical and complicated diagnostic procedures such as angiography, opaque myelography, etc., before a careful and complete history is taken and an examination done. Experience has shown that such diagnostic procedures can reveal localized defects that may be completely asymptomatic so that the attention of the medical examiner is distracted from the real cause of the trouble. Such "red herrings" lead to needless explorations to the embarrassment of the surgeon. When there is available such a simple measure to evaluate the problem from the viewpoint of its extent and degree, it seems pointless to undertake the more complicated tests, at least until there is a reason to do so.

Summary

Pressure neuropathies are common. The typical ones are usually recognized, particularly if the clinician will make a simple investigation as to the circumstances surrounding the beginning of the complaint and examine carefully the part involved. In other cases, especially in atypical and less common situations, definite diagnostic difficulties are encountered. This difficulty stems from the fact that all too often this particular type of pressure neuropathy is new to the physician. Even the experts may have trouble with the unusual or bizarre situations. In such cases the key to the problem is twofold. First it is important for the patient to reenact the situation by which the paralytic state was produced. The second is to use the electromyogram to determine the precise location of the lesion which may lead to the correct diagnosis and thus point the way to successful treatment.

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Seconds count in treating victims of acute injury. A concise and clear plan of management is outlined.

Early Management of the Patient With Multiple Injuries

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Although significant strides have been made in the advancement of safety engineering within the past decades, the problem of the man who is seriously injured at work remains a most important one. According to data published by the Metropolitan Life Insurance Company,[1] approximately 1,800,000 men were injured at work in 1958. Of these injuries, 13,300 were fatal and about 75,000 resulted in permanent physical impairment. The financial burden of these injuries has been estimated at about $3,750,000. They are matters about which all physicians must be concerned.

When the patient who has been seriously injured appears, it is imperative that he receive well-conceived treatment without delay. The management of the patient in the early period following injury is of greatest importance in prevention of unnecessary loss of life or prolonged disability. It is therefore essential that procedures necessary to save the patient's life be second nature to the physician and to others responsible for his care. He must be able to act promptly and surely. He must know that whatever emergency equipment might be necessary is readily available to him and clearly identified.

The management of the patient with multiple injuries should be a cooperative endeavor. The man who assumes initial or over-all responsibility for the patient (usually a general surgeon or a "generalist") must work closely with various specialists who may be required to attend the patient. He must help decide the priority for treatment of the various injuries and must repeatedly reassess the general condition of the patient. In the Cleveland Metropolitan General Hospital a special team has been established to carry out this function in cooperation with members of the speciality services.

Inconspicuous But Serious Trauma

Of prime importance is the recognition of the patient with seemingly minor trauma who may have potentially fatal underlying injury with slowly appearing manifestations. Although a well developed tension pneumothorax is easily recognized, the patient who has sustained a blow to his chest wall, with or without fracture of a rib, may seem deceptively well when first seen. Five or six hours later, however, he may develop serious respiratory embarrassment owing to a small tear in the visceral pleura. The patient who has a bruise over his left lower chest wall may be otherwise asymptomatic and present no physical findings of apparent significance, but he may go into profound shock hours, days or weeks later when the small laceration in his spleen ruptures through the capsule. Head injuries may also present few symptoms and no significant finding early following injury, only to develop bizarre behavior with increasing intracranial pressure and evidence of brain damage much later.

All these lesions may masquerade as trivial injuries when the patient is first seen, and he may be dismissed from the hospital only to suffer avoidable complication and catastrophe later. He may be admitted to a special service

where attention may be so focused on his more obvious injury that a less apparent but potentially dangerous injury is recognized only when the patient goes into shock. This danger is something for which both the specialist caring for the patient and the general surgeon following him must be constantly on the alert.

Obvious Injury

The patient with obvious serious injury presents a somewhat different problem. A number of different organs might be damaged, and each of these may threaten life. It is essential that the physician responsible for the management of the patient have in mind a well conceived and orderly plan of management.

Priority of Treatment

Priority of treatment for the individual patient must be established. This priority should include:

1. Rapid assessment of the patient's general condition.
2. Arrest of any brisk external hemorrhage.
3. Maintenance or restoration of adequate ventilation.
4. Measures to prevent or correct shock.
5. Thorough but expeditious examination.
6. Operative intervention, as part of resuscitation if necessary.

These priorities will be examined more closely.

1. Initial Examination: In the initial rapid appraisal of the patient's status, attempts should be made to detect primarily those disorders of his normal physiology which are incompatible with survival for more than a brief period. The presence or absence of significant external bleeding, respiratory distress, and shock should be determined. These may require correction before time may be taken for a detailed examination.

2. External Hemorrhage: In the event of severe external hemorrhage, control by direct pressure is usually the most effective and least damaging method. Direct clamping with hemostats is satisfactory for smaller vessels, but care should be taken if possible to avoid further injury to major vessels and other important structures. Proximal tourniquets may be required for short periods. This will impose additional hazards, however, and may actually increase bleeding if improperly applied. Direct pressure on the area involved is much to be preferred. Once applied, an arterial tourniquet should not be released until facilities are available for definitive control of the bleeding.

3. Respiratory Embarrassment: Respiratory embarrassment is a frequent complication of trauma and must be corrected immediately.

Airway: First and foremost, any obstruction to the patient's airway must be noted. In unconscious patients, the tongue may fall back to occlude the pharynx and cause asphyxiation. Drawing the tongue forward, adjusting the position of the patient, or inserting an oral or nasopharyngeal airway will overcome this problem. In the event of injury to the face or neck, edema and hemorrhage may cause obstruction of the upper trachea or pharynx. Partial obstruction of this sort may progress very suddenly to complete obstruction, and tracheostomy must be performed before this can occur.

Bronchial secretions may cause significant obstruction to the airway, especially in wounds of the chest and head. With crushing injuries and penetrating wounds of the chest, bronchial secretions may be mixed with blood, compounding the difficulty. It is essential that the tracheobronchial tree be cleared of secretions by effective and repeated aspiration. Tracheostomy may facilitate aspiration and should be done without hesitation when needed.

Ventilation: Inadequate respiratory exchange is an important cause of hypoxia. Shallow respirations are frequently associated with head injury or follow the administration of excessive amounts of morphine. A flail chest wall associated with multiple rib fractures will also prevent adequate exchange. In such instances it may be necessary to assist respirations by means of positive pressure oxygen insufflation with a bag through a properly fitting anesthesia mask or an endotracheal tube. Here again a tracheostomy may be of considerable benefit not only for aspiration of secretions but to reduce the amount of dead space present in the air passages. In the absence of special equipment, artificial respiration should be employed. The most effective method is mouth-to-mouth insufflation with the nares closed, the neck extended, and an airway maintained by means of a finger in the buccal cavity.

Pain: Intercostal pain associated with rib fractures may cause significant limitation of respiratory motion. Procaine block of intercostal nerves will afford dramatic improvement in such instances. The use of morphine should be restricted in the presence of respiratory difficulty.

Pneumothorax: Acute embarrassment of respirations may be caused by the presence of a pneumothorax, either open or closed. An open pneumothorax, or "sucking wound" of the chest, in addition to allowing total collapse of the lung on the affected side, interferes markedly with ventilation of the opposite lung. The open wound must promptly be sealed by means of a large vaseline gauze pressure dressing and the pneumothorax aspirated.

Closed pneumothorax may be caused by blunt

trauma with rupture of the lung, by laceration of the lung on a projecting fragment of broken rib, or by a penetrating wound with laceration of the lung in which the external wound is effectively sealed. Because of the valve-like action of the lacerated lung, tension often develops within the pleural space. This leads to collapse of the ipsilateral lung, shift of the mediastinum to the opposite side, and impairment of ventilation of the opposite lung.

Tension must be relieved by aspiration with provision made for escape of any further accumulation of intrapleural air. An intercostal catheter or a large-bore needle attached to water seal is most effective. In emergency, a flutter-valve made by attaching a moist finger cot with a slit in its distal end to a large-bore needle can be very useful. Inserted into an intercostal space this will allow escape of air and may be easily used while the patient is being transported.

4. Shock: Shock may be defined broadly as "the clinical manifestations of an inadequate volume of circulating blood accompanied by physiologic adjustments of the organism to a progressive discrepancy between the capacity of the arterial tree and the volume of blood available to fill it."[2]

Blood Loss: In the severely injured patient, shock is most commonly associated with acute blood loss. While the source of bleeding may be obvious, it is not always so. Vigorous external hemorrhage may have subsided to a slow ooze by the time the patient presents himself. Significant internal bleeding may occur into the pleural or peritoneal cavities. The amount of blood loss into soft-tissues, especially in association with major fractures, may assume massive proportions. Such blood loss must be compensated by immediate replacement and early control of the source of hemorrhage. Replacement, of course, should consist of whole blood whenever available, although plasma, plasma expanders, or saline may be used in limited quantities if whole blood cannot be readily obtained. Intravenous infusions must be used with some caution in patients with severe pulmonary damage, fat embolism, head injury, or in elderly or debilitated people.

Cardiac Tamponade: A frequently unrecognized and rapidly fatal cause of shock is cardiac tamponade. With immediate proper management, however, the salvage rate for people with wounds of the heart is surprisingly good. Indications of the presence of cardiac tamponade include a lowered arterial blood pressure which is refractory to transfusion, signs of increased venous pressure, a paradoxical pulse, diminished pulse pressure, distant heart sounds, and enlargement of the area of cardiac dullness. Aspiration of the pericardium by inserting an 18 gauge needle just to the left of the xiphoid and angling it acutely upward until the beating ventricle is felt, may establish the diagnosis. Emergency pericardiocentesis and immediate thoracotomy can be life saving and must be undertaken without delay.

Crush: When the circulation to extermities has been occluded by a crushing object and then released with rescue, shock may appear without significant loss of blood. These patients may lose large quantities of fluid into soft tissues and will become hypovolemic with a relatively normal red cell volume and an increased hematocrit. If the hematocrit is greatly elevated, the prognosis is poor. In general, the treatment is somewhat similar to that in severe burns with early use of intravenous plasma and close observation of the urinary output. Intravenous fluids should be carefully administered to establish and maintain diuresis but must be immediately limited if unresponsive oliguria and anuria develop.[3] Irreparably damaged limbs should promptly be amputated.

Pain: The physiologic picture in severely traumatized patients may be profoundly altered by their apprehension and pain. Pain, however, is usually not so severe as one might expect from the magnitude of the injury if the patient is not further traumatized by rough handling. The management of pain, therefore, should be directed largely toward immobilization of the traumatized part and extreme gentleness in moving the patient. The use of morphine should be held to an essential minimum because of its depressant effect. It is also poorly absorbed in the presence of circulatory depression unless administered intravenously. For this reason repeated subcutaneous injections of narcotics must be avoided.

5. Examination of the Patient: The problems with which we have dealt thus far must be treated after only superficial appraisal of the patient. It is important, however, as soon as the patient's urgent physiologic needs have been met, to perform a thorough, although expeditious and gentle, examination. In addition to the usual physical examination including the chest, abdomen, neurological status, and area of obvious trauma, several easily overlooked observations should be emphasized.

(a) The surface of the body should be meticulously inspected for penetrating wounds of which the patient may not be aware. Wounds of the shoulders, back, buttocks, and thighs are commonly missed. Yet they may well be associated with abdominal or thoracic injury, or may be responsible for serious anaerobic infections.

(b) The presence or absence of all major arterial pulses must be ascertained.

(c) Careful examination of sensation and motor power in an injured extremity should be carried out.

(d) Rectal examination is essential in all seriously injured patients, not only to ascertain the presence of rectal injury but to note the presence of blood. Blood in the rectum may signify injury anywhere in the gastrointestinal tract.

(e) All patients with fractures of the pelvis must be assumed to have serious tears of bladder or urethra and must be promptly investigated. Bleeding from the urethra is diagnostic. Immediate urinalysis is essential in all injured patients, especially to note the presence or absence of red blood cells in the urine. The presence of significant numbers of red cells in the urine is diagnostic of injury somewhere in the genitourinary tract. Failure to feel the prostate gland on rectal examination points to severance of the membranous urethra.

(f) In equivocal situations, x-ray findings may be of considerable help. The presence of free air beneath the diaphragm on an upright film is *prima facie* evidence for rupture of the stomach or intestine. Elevation of the left leaf of the diaphragm, increased density in the left upper quadrant, indentation and serration of the greater curvature of the stomach, or depression of the splenic flexure of the colon may suggest rupture of the spleen. Retroperitoneal gas may indicate the presence of rupture of an extraperitoneal portion of the duodenum or colon. Emergency cystography and intravenous pyelography may be indicated to help detect injury to the urinary tract. Early abdominal roentgenograms may be especially helpful to compare with later films, which may show change in fluid or gas patterns or in visceral contours.

(g) Most important in the examination of patients with major injury is to avoid concentrating on an obvious injury to the extent that less apparent but serious injuries are overlooked.

6. Surgical Intervention: Last but not least on a priority list for the patient with multiple injuries is consideration of surgery. There are many instances where formal operative procedures must be considered an integral part of resuscitation. To wait for the patient with continued bleeding from his ruptured spleen or torn internal mammary artery to "stabilize" is to invite disaster. Ideally the optimum time for exploration is when the patient's blood pressure begins to rise following the resuscitative measures outlined above. With severe bleeding, however, this may not occur, and every minute of delay will prejudice the patient's opportunity to recover. If one waits until a normal blood pressure has been obtained, vigorous hemorrhage may begin again. The second episode of shock so engendered may be much more difficult to combat than the first.

An equally important part of resuscitation may be surgery to prevent infection or to curb infection once it has become established. Extensive extravasation of urine from a ruptured bladder, retroperitoneal dissection from a lacerated duodenum, or contamination of the peritoneum from a perforated colon will lead to continued invasive infection. Resuscitation may be impossible until these factors have been brought under control.

When intra-abdominal injury is seriously suspected, early operation should be undertaken. The occasional negative exploration is more than justified in order to avoid the serious or fatal complications of delay.

Summary

Priority of treatment for the individual patient with multiple injuries should be established. The importance of such a priority should be stressed in hospital teaching programs.

Such a priority should include:

1. Rapid assessment of the patient's condition.
2. Arrest of brisk external hemorrhage, by local pressure if possible.
3. Maintenance or restoration of adequate ventilation, including adjustment of airway, aspiration of secretions, administration of oxygen, tracheostomy when indicated, and treatment of pneumothorax.
4. Measures to prevent or correct shock.
5. Thorough but expeditious examination.
6. Operative intervention, as part of resuscitation if necessary.

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Early use of specialized rehabilitation facilities can save manpower waste and costly litigation.

MODERN REHABILITATION

As Seen by an Industrial Physician

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A report by the New York division of the Office of Vocational Rehabilitation during the fiscal year 1953-1954 indicates that 655 requests for lump-sum settlements of compensation cases involving permanent injury were made. In less than half of these (309) the patients had been or were being rehabilitated. The probable reasons for this relatively low percentage were given as follows: (a) lack of motivation, (b) ignorance or venality of doctors, lawyers, and workmen under present medical care provisions and claims procedures, (c) lack of adequate authority for compensation boards to supervise medical care and failure by the boards to exercise what authority they have, (d) lack of facilities for rehabilitation and shortage of trained personnel.

Considering these four points, it is not hard to explain why it has been difficult to get this third phase of medicine — rehabilitation — out of the discussion stage.[1]

Motivation

Recently, Jones & Laughlin joined with two other industrial corporations and a rehabilitation center in a study of our disabling injury and illness experiences. The goal is to test our past and current practices with a view toward reducing disability and increasing productiveness. Already we are recognizing certain truisms — that not all disabilities can be reversed or even prevented; that it is advantageous to bring the rehabilitation consultants into the case management shortly after treatment for injury has been started; that it is necessary to place more emphasis on the psychosocial aspects of all disability problems; that early rehabilitation on the job is usually adequate and desirable provided there is real understanding of the type of job to which the patient is going; and that energetic follow-up practices are required to prevent stagnation, discouragement, and fixing of a disability to a greater degree than necessary.

The rehabilitation literature abounds with pleas for early referral. Dr. Rusk is quoted as saying: "Early reference is the greatest problem we have in the rehabilitation program today. We get patients who have gone through a cycle just as real and just as definite as that in tertian malaria. The cycle runs through physical neglect, emotional trauma, and vocational sterilization."[1]

Litigation

One of the recognized barriers to early rehabilitation is the matter of secondary gain. It

Read at the Eighth World Congress, International Society for The Welfare of Cripples, New York, Aug. 28-Sept. 2, 1960. Dr. Lauer was formerly Medical Director of Jones & Laughlin Steel Corp., Pittsburgh.

is somewhat difficult to retain the features of just compensation without impairing motivation for rehabilitation. The conflict between compensation payments and medical care and rehabilitation will be present as long as the primary emphasis of the compensation set-up is upon monetary payments. The problem is how to minimize this conflict.

We are all well aware of the psychological trauma produced by litigation. At times this is one of the major barriers to early rehabilitation.

The province of Ontario is often credited with an outstanding workmen's compensation system. Commissioner Steele of their board recently wrote: "The Ontario plan of compensation, based on a clinical rather than on an adversary approach, is, in effect, a large-scale rehabilitation effort. The individual physician plays a vital role in a team effort to return injured employees to the job quickly with minimal disability. Our concept of rehabilitation includes skillful medical attention at the outset, along with all the skills, modalities, and techniques available to restore the patients to their pre-accident state. From our experience over the years, we are convinced that this approach is more humane to the injured workman and less costly to the employer. Those groups of the community most vitally interested — organized labor, employers, and the medical profession — endorse our rehabilitation-compensation system."[2]

Many states have lump-sum settlement provisions in their workmen's compensation laws. Administratively, it may be desirable, but from the standpoint of the workman undergoing rehabilitation and correction of his disability, it is of dubious value. Before the settlement is made, a workman does not wish to undergo rehabilitation for fear of jeopardizing the amount of the settlement. After the settlement, the employer, or his insurance carrier, is under no further obligation since the case is closed.

Other factors that militate either for or against rehabilitation are matters dealing with the limitation in time and the amount of medical care and compensation according to the law. Limited liability is really one of the inherent factors in a workmen's compensation system, although a number of states have written into their compensation law the requirement of the employer, or his insurance carrier, to provide hospital and medical services for a lifetime. Unless means of rehabilitation can be applied, costs on individual cases may run to $100,000 or more.

Facilities

As physicians and surgeons, we are still in a phase where we are somewhat hesitant to call upon the techniques and personnel of rehabilitation. But as the demonstrated value of modern rehabilitation imposes its wonders upon us, such resistance will evaporate. The evolution will progress proportionately to the time we take to visit modern rehabilitation centers, to observe their work, to study their methods, to understand their philosophy, and to personalize it in practice.

There still exists a paucity of rehabilitation centers in the United States. Such centers are not in competition with the physical medicine departments of our general hospitals. Hospitals are eager to avoid the long stay occasioned by chronic disease or severe injury cases. It is economically imperative that our hospitals reduce the long stay — the warehousing of the sick and the injured.

The successful rehabilitation of one injured workman is in itself a stimulating factor for the others. Success is the key to future successes. The competition of like groups of workmen living, playing, exercising, and working together often has the effect of reducing pain and fear. Confidence leads to restored muscle power and restored attitudes of happiness.

We all agree that it would be futile to attempt to make rehabilitation compulsory.[3]

Placement

Two major objections are frequently put forward for refusing to hire handicapped workers. First, the impaired person represents a greater liability risk to the employer. Second, insurance rates are increased.

In an effort to overcome the first of these objections, the legislatures of many states have passed second injury statutes with the purpose of encouraging the employment or re-employment of the handicapped. In principle, this is good; in practice, it does not always work out so well.

Generally, the first requirement for qualification under the second injury statute is that the employee must have already lost or lost the use of a limb or had some other permanent physical impairment. Second, the employer must have been fully aware of this physical impairment. Third, the permanent disability attributable to both the original and the second injury must be more serious than that caused by the second injury alone. There have been various legal interpretations read into these general requirements that have interfered with the good intentions of the code.

Second injury funds lose effectiveness when there is a question of aggravation of a pre-existing disease or defect for which compensation may or may not have been paid.[4] Employment of persons with progressive disabilities —

neuropsychiatric, cardiac, pulmonary diseases, arthritis, and deformities or anomalies of the spinal column — is particularly hampered.

It is discouraging to have legal interpretations so classify diseases that acute episodes which by chance may occur while an individual is engaged in his regular work are attributed to the work. This kind of chicanery only interferes with understanding of disease, and with proper acceptance of persons with such diseases into useful employed society.

We could all agree that continuing physiologic research into energy demands of the various job types, and the reactions in health and disease to them will be most useful in improving our basis to employ handicapped persons. It will aid in selectively placing them in suitable jobs. The interest in returning victims with various kinds of heart disease to useful employment has encouraged Ford and Hellerstein to do extensive studies in estimating physiologic tolerances in many job types.[5]

The second major objection to hiring the impaired person frequently raised by employers is that an increase in insurance premiums would result. This would probably be true if there is no selectivity exercised in job placement.[6]

The key to the hiring of new impaired workers is proper job placement. This cannot be too strongly emphasized. When it is carried out, there is an abundance of statistical evidence and reports to indicate that the physically impaired workman is as safe as or safer than his so-called normal counterpart. Trade unions recognize the needs and values for such practices but at local levels are slow to allow them to be put into practice if interference with the seniority structure appears likely.[7]

In the final consideration of the question, a handicapped person is not really handicapped if matched by ability to the job he is to do.

Summary and Conclusions

Rehabilitation is a multidisciplined and multifaceted aspect of health and disease. It has grown markedly in the past two generations. One can almost measure a civilization's progress by its willingness to restore the sick and the maimed.

Rehabilitation is not only economically sound today but is becoming an economic necessity. We all need to learn much more of the many cases amenable to rehabilitation; to know what consultation to seek; when to seek it, and how to use it, whether for physical or mental reasons or both.

The pressures of society, through law, are demanding better restoration, better medical care, better rehabilitation. We in the health professions must be alert to these demands — we must lead them into constructive areas and keep them economically feasible.

Those of us in industry whose primary concern is with the occupationally connected health problems of our working population must constantly re-examine our methods and our attitudes toward rehabilitation. I believe all of us need to do a more complete and effective job in the psychosocial area of rehabilitation.

We must counsel with the injured workman right from the start of his treatment by answering his fears and doubts, telling him what is to be done for him on a program of medical treatment and rehabilitation, what he can reasonably expect and when; and what is planned for him upon his return to productive employment. If we could just place ourselves in his position, I think all the answers to the questions would readily present themselves.

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REHABILITATION
Of Workers Addicted to Alcohol

The Medical Division of the du Pont Company has always been somewhat amazed that so many people are surprised that we have alcoholics in our employ; that we treat the problem of alcholism as a disease; that we have a plan to bring the alcoholic back to sobriety and that our doctors are trained and urged to bring these problems into the open when they occur.

There should be no surprise at all, for, as the late Dr. George Gehrmann, our former Medical Director, repeated over and over again, "Where you have people and alcohol, you have alcoholism." We have people in our employ, and we have studied the problem known to have existed for the past 16 years, since we have had a program for the rehabilitation of the alcoholic workers among an average annual employment of 87,131 people. That alcohol is readily available to all is without dispute. Therefore, we have both raw materials for the end product.

That alcoholism is treated as a disease is, in our opinion, based on sound rationale. Let us quote the definition of "disease" from the dictionary: "In general, any departure from a state of health; an illness or a sickness. More specifically, a definite morbid process having a characteristic train of symptoms. It may affect the whole body or any of its parts, and its etiology, pathology, and prognosis may be known or unknown."

Certainly, the idea that alcoholism is a disease is quite compatible with this definition. However, contrary to some other diseases, the disease alcoholism is under the control of the will in that one does not have to drink if he makes up his mind to not do so. But this does not alter the fact that the great drive to drink is present. And, be it psychological, psychiatric, biochemical, or some other kind of drive, it is abnormal. Alcoholism fits the definition of disease more adequately than some others accepted as such but based on less fulfillment of the definition.

Read at the Eighth World Congress, International Society for the Welfare of Cripples, New York, Aug. 28-Sept. 2, 1960.

Our concept of alcoholism as a disease is that, contrary to many other diseases, alcoholism is self-induced. We must assume that one who cannot drink does not have to, or at least he should not. The cause of alcoholism is known. That one has the power to refrain from drinking whenever he develops the will to do so in spite of the compulsion to drink is firmly attested by the thousands and thousands of members of Alcoholics Anonymous. Thus, the disease is preventable.

The alcoholic has been variously described as infantile, recessive, neurotic, selfish, irresponsible, and even homosexual. He has been accused of being a derelict and of lacking in moral substance. He has been linked to various and sundry psychiatric disorders and patterns. It has been our experience that the alcoholic, when sober, is just like the sober nonalcoholic. He is the person working or living next to you. When he is sober and not drinking at all, he is generally indistinguishable by personality, appearance, mannerism, or character from the other people about the office or home. He is not, in our opinion, a type, a character, or a particular build, but just a person. When he is drinking, however, he is apprehensive, anxious, jittery, perspiring, suspicious, easily swayed, and weak. The end degree of this is well known to you.

Our plan was inaugurated by Dr. G. H. Gehrmann in 1944. It is a simple plan, and fundamentally one to teach all employees to recognize alcoholism and, when discovered, to bring it into the open rather than to "sweep it under the carpet." It is dealt with honestly and the employee is given the solid and clear offer to have it treated both fairly and honorably. One would not conceivably want to conceal a fellow employee who had syphilis or diabetes. Why then would he want to conceal a fellow employee who has the disease alcoholism? The ravages and sequelae of alcoholism are no less than the others.

The alcoholic worker is given every opportunity

to lick his problem and he is fired only if he is unwilling to overcome it after due consideration of time and effort. This applies to any disease producing disability, curable or amenable to therapy, to the extent that the individual cannot do his job properly or safely. Further, one who chooses not to overcome any progressive disorder is a liability, not only to himself but to his fellow employees — and we have a responsibility to all employees.

We attempt to ferret out the cases and to bring them to the fore. They are all given a satisfactory time to rectify the situation after orientation by the doctor as to our concepts of the problem as a disease; then they have a discussion with our adviser on alcoholism, or some member of Alcoholics Anonymous, usually another employee of the same sex and often in the same department, and finally they are supervised. The amount of time given to straighten out, and whether a leave is given, will vary with the worker's age, years of service, previous opportunities given, nearness to retirement age, complications of the disease, whether these complications are permanent or temporary, the employee's attitude toward his problem and his sincerity of effort. In the absence of medical complications, it is rare that more than two weeks away from work are necessary, and then only for institutional treatment. More often, no lost time from work at all is desirable. Experience has taught us that alcoholics do better with surveillance on the job. This is the same experience noted by the military psychiatrists, as well as by those experienced in rehabilitation.

We have found that close cooperation with the program of Alcoholics Anonymous has given us by far our best results. We have also learned from experience that, when our psychiatrists work closely with this program, the results are further enhanced. We have a fulltime employee, himself a "recovered" alcoholic and a member of Alcoholics Anonymous, who works under the guidance of our psychiatrists, gives talks to plant groups and assists in the handling of these cases. This program is effective and we know of no other program to equal it.

As for talking to a man about his drinking, this is not the job for the inexperienced — it is one of the hardest jobs in the world. Many cannot do it. And it is rare that a man comes to the conclusion all by himself that he has a drinking problem. When it is adequately explained to him, the victim is often relieved to be shown the way out of his dilemma.

In a survey of our company, we ascertained that, of an average company population of 87,131 employees, we have uncovered 950 alcoholics up to the present time. This is a prevalence rate of 1.09%. We are confident that an undetermined number has not yet been reached or uncovered. And we, like others, have not yet solved the many facets of the hidden problem of alcoholism. Our job in this respect is certainly not done, nor can we foresee the time when it will be. However, among the known 950 alcoholics, we found that we have rehabilitated 66% of them; we have helped and improved, but not totally recovered, another 23%, and the remaining 11% are either unchanged or their present status is uncertain.

That our doctors are trained and urged to bring this problem into the open is just part and parcel of the practice of good medicine. No good doctor, be he a general practitioner or an industrial physician, can fail to appreciate not only these facts, but the so-called hidden aspects of drinking, a staggering problem not yet adequately explored. Although I do not have the statistics, I can assure you that the hidden aspects of alcoholism far outweigh the obvious aspects. The unhappy marriages, the high divorce rates, the suicides, the concomitant psychiatric or social problems, the toll of liver disease and cirrhosis, the flare-up of peptic ulcers, the "obscure" convulsions, the nutritional deficiencies, and the recurrent breakdowns of such chronic diseases as tuberculosis, respiratory diseases, arthritis and others, are so closely wrapped with and allied to the problem of alcoholism in many instances that if these could be calculated and appraised, for the alcoholic as well as industry and his family, the resulting figures would certainly be staggering.

In consideration of all of these, how then can any physician say that he is not interested in the problem of alcoholism? The problem of alcoholism is the problem of people, and the problems of people are the province of the doctor.

The literature pretty well establishes proof that the alcoholic loses about 22 working days a year more than the average employee; he loses two more days for other illnesses than the other employees; he has twice as many accidents as other employees; and his life expectancy is 12 years less than that of the nonalcoholic. How then can an employer, friend and fellow employee, or we in the Medical Division turn our backs on such a problem?

The controlled alcoholic is a good employee and he is good for other employees. He is, in brief, worth saving. Our program is effective. There is nothing complicated about it. No large outlay of funds is necessary for it. We would certainly commend such a program to any other organization which employs people and which wishes to face a problem which is almost certainly there.

NEW CONCEPTS OF INDUSTRIAL DUST DISEASES

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All our lives, with nearly every breath, we inhale dust. In general, city air contains over twice as much dust as country air and the working atmosphere within many industries contains tremendous amounts of dust. Most dusts are innocuous although a few dusts have varying capacities to produce disease (Table I).

How the Lung Handles Dust

Ordinarily, dust particles over 10 μ in diameter have such mass that they settle out of the air rather quickly. The few particles inhaled are trapped in the nose and upper respiratory tract. The sharp angulations, moist surfaces and coarse hairs of the nasal passages are designed to catch the larger dust particles in the inhaled air. Mostly dust particles 10-5 μ in size are removed from the air in the upper respiratory tract while dust particles 5-3 μ in size are deposited in the mid-respiratory tract. Dust particles 3-1 μ in size may be deposited directly in the pulmonary alveoli. Dust particles 1-0.1 μ, with decreasing size, are deposited less readily in the alveoli for they do not settle out. Finally, dust particles less than 0.1 μ in size have such small mass that they are in Brownian movement, moving in and out of the alveoli with the air. However, it is now believed that many of these particles do deposit upon alveolar walls through impaction by Brownian movement and by being caught by movement of the alveolar walls.

The fate of deposited particles: Dust particles deposited in respiratory tract above the alveoli are swept upward by the ever-present cilia. The entire respiratory tract to the terminal respiratory bronchioles is covered by a thin layer of fluid and minute cilia that move in unison whipping upward deposited dust particles, mucus, and fluid at a rate of about 1 cm per hour in the bronchioles, increasing to a rate of perhaps 3 cm per hour in the trachea. Unless the dust particle is soluble, it is returned to the pharynx where it is expectorated or swallowed.

Particles deposited in alveoli: If the particles are soluble, they may be absorbed directly into the blood stream. Some nonphagocytized insoluble particles move out of the alveoli in the surface fluid passing into the atria. Here they move either into a lymphatic or they are picked up by the cilia and carried up the bronchial tree. In addition some of the smaller dust particles penetrate directly through the alveolar wall into the peribronchial sheathes, lodging frequently about the pulmonary arterioles. Phagocytic cells, mostly mononuclear histiocytes and undifferentiated mesenchymal cells, abound in the thin alveolar wall about the pulmonary capillaries. The presence of dust particles stimulates these cells to activity. These phagocytes engulf dust particles and then by ameboid action move out of the alveolus and enter into a lymphatic, or move directly through the alveolar wall into the peribronchial spaces, or proceed out of the alveolus into a bronchiole where the cilia sweep them up the bronchial tree. The majority of dust particles undoubtedly are returned up the bronchial tree.

Comment: Two additional points regarding the deposition and handling of dust particles in the lung require mention.

Mouth breathing, since it shorts out the nose and nasopharynx, apparently markedly increases deposition of larger dust particles in the mid and lower respiratory tract.

Lastly, unless they are overloaded by dust particles and dust laden phagocytes, little or no dust accumulates in the lymphatic vessels and only small amounts in the peribronchiolar sheathes. Most of the dust particles and dust laden phagocytes entering the lymphatic system of the lungs are deposited in the bronchial and hilar lymph glands, although it is possible that some dust may traverse the thoracic duct and reach the systemic circulation.

The Pulmonary Response to Mineral Dusts

Storage of insoluble dust in the lung during life is in direct proportion to the concentration and duration of dust inhaled and in inverse proportion to the size of the dust particles. Excessive storage of dust in the lungs results from the inhalation of abnormal amounts of small dust particles, particularly those less than 10 μ in diameter over a sufficient period of time. In the face of excessive deposition of dust, the self-cleansing mechanism becomes overloaded, the surfaces of the alveoli become covered by dust particles and many dust laden phagocytes. A great number of such phagocytes collect in the perivascular and peribronchial sheathes, in the lymphatic vessels, and in the foci of lymphoid tissues about the bifurcations of the bronchioles.

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Particularly when the bronchial and hilar lymph nodes are overburdened, both these glands and the lymphatics become overdistended and clogged. The subpleural lymph spaces equally become dilated and clogged by dust laden phagocytes.

Many dust laden phagocytes die or become fixed, depositing dust particles all along the lymphatics and in the perivascular and peribronchial sheathes. At this stage, and it matters not what the insoluble dust is, throughout the

TABLE I*

PULMONARY RESPONSES TO CHIEF INDUSTRIAL MINERAL DUSTS (IN PURE STATE)

Technical Name	Physical Character	Chemical Formula	Tissue Response	Possible Entity
FREE SILICA				
Quartz	Crystalline	SiO_2	Proliferative	Silicosis
Chalcedony	Cryptocrystalline	SiO_2	Proliferative	Silicosis
Tripoli	Cryptocrystalline	SiO_2	Proliferative	Silicosis or none
Chert	Cryptocrystalline	SiO_2	Proliferative	Silicosis
Flint	Cryptocrystalline	SiO_2	Proliferative	Silicosis
Agate	Cryptocrystalline	SiO_2	Proliferative	Silicosis
Tridymite	Crystalline	SiO_2	Proliferative	Silicosis
Cristobalite	Crystalline	SiO_2	Proliferative	Silicosis
Diatomaceous earth	Amorphous	SiO_2	Foreign body reaction	A minor pneumoconiosis
Diatomite-calcined diatomaceous earth	15-60% Crystalline (as crystobalite)	SiO	Proliferative	Diatomite pneumoconiosis (silicosis)
Taconite	Crystalline	Fe_2O_3 & SiO_2	Proliferative	Taconite pneumoconiosis or siderosilicosis
Synthetic silicas (e.g., Degussa)	Amorphous	SiO_2	Varies—some proliferative depending on particle size	Emphysema in animals, bronchitis, bronchiolitis
SILICATES				
Asbestos				
Chrysotile	Fibrous	$3MgO \cdot 2SiO_2 \cdot 2H_2O$	Proliferative	Asbestosis
Amphibole Group	Fibrous	$2CaO \cdot 5MgO \cdot 8SiO_2$ (Variations)	Proliferative	Asbestosis
Commercial talcs	Fibrous and/or foliated			
Talc	Fibrous and/or foliated	$3MgO \cdot 4SiO_2 \cdot H_2O$	Proliferative to fibrous form	Talcosis
Tremolite	Fibrous and/or foliated	$Ca_2Mg_5(OH)_2 \cdot (Si_4O_{11})_2$	Proliferative to fibrous form	Talcosis
Anthophyllite	Fibrous	$(Mg,Fe)SiO_3$	Proliferative	Talcosis
Mica				
Muscovite (sericite)	Crystalline	$K_2O \cdot 3Al_2O_3 \cdot 6SiO_2 \cdot 2H_2O$	Foreign body reaction	Minor silicate pneumoconiosis
Biotite	Crystalline	$H_2K(Mg,Fe)_2 \cdot Al(SiO_4)_2$	Foreign body reaction	Minor silicate pneumoconiosis
Vermiculites	Plates	Varied hydrated silicates	Foreign body reaction	Minor silicate pneumoconiosis
Clays				
Kaolinite	Crystalline	$Al_2O_3 \cdot 2SiO_2 \cdot 2H_2O$	Foreign body reaction	Minor silicate pneumoconiosis
Fuller's earth	Crystalline	$Al_2(SiO) \cdot H_2O$ (variable)	Foreign body reaction or diffuse fibrosis	Minor silicate pneumoconiosis
Montmorillonite (bentonite)	Crystalline	$(Mg,Ca)O \cdot Al_2O_3 \cdot 5SiO_2 \cdot nH_2O$	Foreign body reaction	Minor silicate pneumoconiosis
Feldspar				
Orthoclase	Crystalline	$KAlSi_3O_8$	Foreign body reaction	Minor silicate pneumoconiosis
Microcline	Crystalline	$KAlSi_3O_8$	Foreign body reaction	Minor silicate pneumoconiosis
Albite	Crystalline	$NaAlSi_3O_4$	Foreign body reaction	Minor silicate pneumoconiosis
CARBONATES				
Calcite (chalk, limestone, marble)	Crystalline	$CaCO_3$	Inert	None—(chalicosis)
Magnesite	Crystalline	$MgCO_3$	Inert	None (chalicosis)
Dolomite	Crystalline	$CaMg(CO_3)_2$	Inert	None (chalicosis)
OTHER MINERALS				
Hematite	Crystalline	Fe_2O_3	Inert	Siderosis
Magnetite	Crystalline	$FeO \cdot Fe_2O_3$	Inert	Siderosis
Cassiterite (tin)	Crystalline	SnO_2	Inert	Stannosis
Barite	Crystalline	$BaSO_4$	Inert	Baritosis
Titanium dioxide (rutile)	Crystalline	TiO_2	Inert	Titanosis
Silicon carbide (carborundum)	Crystalline	SiC	Inert	A minor pneumoconiosis
Aluminum oxide (artificial abrasive, synthetic emery, corundum)	Crystalline	Al_2O_3	Inert	A minor pneumoconiosis
Garnet (almandite)	Crystalline	$Fe_3Al_2(SiO_4)_3$	Inert	Minor silicate (pneumoconiosis)
Graphite (plumbago, black lead)	Crystalline	Pure carbon	Inert	Graphosis
Soot	Amorphous	Soot—carbon	Foreign body reaction	Anthracosis
Coal	Amorphous	Carbon	Foreign body reaction and fibrosis	Coal miners' pneumoconiosis
Portland cement	Amorphous	2 & $3\ CaO \cdot SiO_2$ $3CaO \cdot Al_2O_3$ $4CaO \cdot Al_2O_3 \cdot Fe_2O_3$	Inert or absorbed	None or minor pneumoconiosis
Calcium sulphate (gypsum)	Crystalline	$CaSO_4$	Inert	None

*Taken from Johnstone & Miller, *Occupational Diseases and Industrial Medicine*, W. B. Saunders Co., Philadelphia, 1960.

lung the excessive accumulation of dust particles and cells gives the appearance of a lace work of linear dust deposits with focal accumulations of dust particles in lymphoid foci. Thus is a pneumoconiosis developed. Whether impaired pulmonary function ultimately results depends upon two factors, the development of proliferative fibrosis and the magnitude of damage inflicted upon the bronchioles, alveoli and the pulmonary blood vessels. With severe damage, emphysema may result adding to the disability.

Silica-nodular silicosis: Crystalline silicon dioxide particles from 3 to perhaps 0.1 microns in size when stored in excessive amounts in the lungs over a period of time, usually years, characteristically produce fibrous nodules that have been characterized as "dust granulomas." These nodules develop by an unusual fibrotic process. Basically each little nodule is believed to evolve from a special "irritant type" reaction although a body of evidence is growing for the theory of an immunological tissue response being responsible for the formation of these minute nodules. Previously it has been believed that clusters of dust laden phagocytes and free silica particles lodged in the small aggregations of lymphoid tissue in the subpleural lymphatics, or as collections of dust particles about a pulmonary arteriole stimulated the characteristic fibrotic tissue proliferation.

Gross, working with rats, using special techniques, has shown that most silicotic nodules orginate in or on the alveolar walls and that the collagen fibers spring from the cells of the alveolar walls. In either event, the nodule develops by a peculiar fibrotic process in which fibrous tissue is laid in concentric rings about this central core or focus of silica particles as if to wall it off, enveloping it by layers of fibrous tissue like the layers of an onion. There result macroscopic, round, hard, discrete, solid fibrous nodules 2 to 4 mm and occasionally even 6 mm in size. Upon reaching this size, these individual nodules will become stationary if no further exposure to silica occurs. Such nodular silicotic lesions may be distributed fairly uniformly throughout the lungs although irregular distributions sometimes occur due to previous lung disease where old scars and deformities are apt to trap more dust particles. Also, sometimes closely contiguous nodules during their development coalesce and unite, forming a common capsule and producing small fused or conglomerate nodules. Obviously, the many individual alveoli immediately about the nodules are distorted, compressed or collapsed. Pressure on blood vessels, particularly the pulmonary arterioles, render other alveoli avascular and nonfunctional. The resulting distortion with some breakdown of alveolar septa immediately about such nodules, produces minute foci of emphysema. Yet the majority of the intervening alveoli are normal. Thus, although there occur small amounts of alveolar collapse and destruction, so great is the functional reserve capacity of the lung that little or no demonstrable impairment of pulmonary function results.

Silica-non-nodular silicosis: Minute or ultramicroscopic silica particles, sometimes fume in character, produce a more immediate reaction. Little is known regarding the specific action of such particles save that some may produce a rather immediate intense irritant reaction in the bronchioles and alveoli resulting in an acute bronchiolitis that soon is followed by diffuse fibrosis without nodulation.

Shaver's disease is an example of a special form of non-nodular diffuse fibrotic silicosis with ultramicroscopic free silicon dioxide particles being the etiologic agent. Although silica is present in crude bauxite only as a 5 to 7% impurity, yet crystalline SiO_2 comprises 30 to 40% of the furnace fume with a particle size below 0.1 μ. Vorwald has demonstrated that other submicron silica dusts are capable of producing diffuse fibrosis without nodulation.

Silicates: Of the silicates, only asbestos and talc when inhaled in pure form are capable of causing a true diffuse fibrotic reaction and this is probably due to mechanical stimulation of the tissues by the hard asbestos or talc fibers. The other silicates in pure form and in the absence of free crystalline silicon dioxide, the micas, clays and feldspars, produce only a mild foreign body reticular type reaction characteristic of the silicatoses.

Carbonates: These materials are inert and in pure form result in no tissue proliferation or changes even when inhaled in large amounts over a long period of time.

Other minerals: The iron, tin, barium, and titanium oxides when inhaled in pure form in excessive amounts cause no tissue reactions. However, the collection of these dust particles in foci result in nodular, often dense opacities as seen upon chest x-ray. Nevertheless, tissue sections reveal no tissue reaction about these focal collections of metallic particles. Coal dust, on the other hand, may produce fibrosis and coal miners' pneumoconiosis.

The Classification of the Pneumoconioses

Table II presents a grouping of the pneumoconioses upon a pathological basis into major, minor, and benign pneumoconioses — a logical workable classification. The major pneumoconioses consist of classical nodular silicosis, the non-nodular diffuse fibrotic type of silicosis,

TABLE II**
THE PNEUMOCONIOSES

Entity	Dust
The Major Pneumoconioses	
Nodular Silicosis	Free Crystalline SiO_2
Non Nodular Silicosis	
Silica Pneumonitis	Ultra microscopic-crystalline SiO_2
Diatomite Pneumoconiosis	Calcined diatomite-crystalline SiO_2
Shaver's Disease	SiO_2—as a fume
Asbestosis	Silicate—3 $MgO \cdot 2SiO_2 \cdot 2H_2O$
Talcosis	Silicate—3 $MgO \cdot 4SiO_2 \cdot H_2O$
Coal Miners' Pneumoconiosis	Coal dust—carbon
The Minor Pneumoconioses	
Anthracosis	Soot and carbon
Diatomaceous Earth Pneumoconiosis	Amorphous SiO_2
Silicatosis	
Micas (pure)*	More or less complex silicates with silica bound in molecule
Clays (pure)*	
Feldspars (pure)*	
Vegetable Dust Pneumoconioses	
Mill Fever	Various organic dusts
Byssinosis	Cotton
Bagassosis	Mouldy sugar cane
Farmer's Lung	Mouldy hay, straw
Grain Asthma	Grain
Tamarind Asthma	Tamarind Seed
Weavers' Cough	Mouldy cotton yarn
The Benign Pneumoconioses	
Baritosis	Barium sulfate—$BaSO_4$ (pure)*
	Barium oxide—BaO (pure)*
Siderosis	Ferric oxide—Fe_2O_3 (pure)*
Stannosis	Stannic oxide—SnO_2 (pure)*
Titanosis	Titanium dioxide—TiO_2 (pure)*
Graphosis	Graphite—carbon (pure)*
Chalicosis	
Limestone, marble, cement	Calcium salts (pure)*

*The word "pure" is used to denote absence of silica.

**Taken from Johnstone & Miller, *Occupational Diseases and Industrial Medicine*, W. B. Saunders Co., Philadelphia, 1960.

asbestosis, talcosis and miner's pneumoconiosis.

The minor pneumoconioses consist of diatomaceous earth pneumoconiosis from amorphous silica, and the silicatoses from pure micas, clays and feldspars. In addition, the organic or vegetable dusts causing pneumoconioses also are grouped under the minor pneumoconioses, although at times temporary severe disabling diseases may be produced. Still, in time with removal from the offending vegetable dust, complete recovery is to be expected with little or no physical or radiological evidence of the previous pneumoconiosis remaining.

The third group are the benign pneumoconioses. Here are those heavy metal oxides or sulfates such as tin, iron, barium and titanium that provoke no reaction when deposited in pulmonary tissue. Although the x-ray film may be startling in that it reveals innumerable nodular densities, yet about these nodular deposits of metallic dust particles, there is no tissue reaction to be found on histological sections. In addition graphite, pure carbon, and pure marble, cement and limestone dusts are also inert and incapable of producing tissue reaction even when deposited in the lung in large quantities.

Summary

Careful study of the various mineral dusts, familiarizing oneself with the technical name, physical character and chemical formula, the type of pulmonary tissue response and hence the possible entity, is of great help in creating an orderly understanding of the pneumoconioses (Table I). The grouping of pneumoconioses in major, minor, and benign groups upon a pathological basis facilitates understanding of these entities (Table II).

Free crystalline SiO_2 is capable of causing two types of disease both properly called silicosis. First, particles 3 μ to 0.1 μ in size can produce classical nodular silicosis. Second, ultramicroscopic sized particles may cause a non-nodular silicosis characterized by production of diffuse fibrosis. It was shown that silicotic nodules may arise directly upon and from the alveolar wall in response to collections of silica particles adhering to the alveolar wall. Quite possibly non-nodular silicotic fibrosis may arise in the same manner, however as yet this is conjecture.

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Chronic Carbon Monoxide Poisoning

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"The question of the existence or not of chronic carbon monoxide poisoning has been much discussed and is not yet settled. In any case, one should be very cautious before assigning the signs observed to the action of carbon monoxide because the connection is often far from clear. On the other hand some authorities consider the action of small doses of carbon monoxide (CO) cannot be without effect on the organism not only on the blood and the blood forming tissues but also on the metabolism. They consider that 80% of the cases of poisoning by CO remain unrecognized because the sufferers complain of indefinite symptoms that remain incurable and cannot be diagnosed." The above statement appeared in an International Labor Office publication in 1930.[1]

Hamilton and Johnstone, in a 1945 publication[2] state, "Chronic CO poisoning still is a field of controversy, for although there is general agreement that it is an actual industrial disease there is little agreement as to the character and the severity of the symptoms, which may be caused by long exposure to low concentrations of this gas. It is an extremely important subject, for many thousands of workers are exposed in this way. That there is no accumulation of CO in the body is beyond question. It disappears from the blood, usually within minutes or a few hours. Damage is to be attributed to the cumulative effect of repeated doses."

Sax[3] cites the effects of chronic exposure to CO as described in Scandinavian literature. Auditory disturbances and contraction of visual fields have been demonstrated as well as glycosuria and heart irregularities.

Katz,[4] reviews 41 cases of chronic CO poisoning seen in Swift Current, Saskatchewan, between 1954 and 1957. Presenting symptoms were headache, anorexia, dyspepsia, weakness and dizziness, singly or in combinations. The finding of carboxyhemoglobin (COHb) level of 10% or more plus the relief of symptoms when the patient is removed from exposure confirms the diagnosis.

In contrast to the above, Hunter, a British physician,[5] says that CO is not a cumulative poison and that in pure air, with a sufficient volume of breathing, small amounts of this gas are removed from the body. There is, therefore, no such condition as chronic CO poisoning.

In the course of a number of industrial hygiene surveys performed by the Environmental Research Laboratory, numerous complaints have been investigated involving occupational exposures to CO. In most cases, atmospheric levels of the contaminant were just at or slightly below the threshold limit value for CO of 100 parts per million (ppm). There were times, however, throughout the working day, particularly in automotive repair garages, when personnel were subjected to concentrations ranging from 200 to 400 ppm. These episodes usually lasted from three to 10 minutes and occurred approximately four to six times a day. Usual concentrations of CO ranged from 30 to 100 ppm.

Many of the employees involved in these investigations complained of symptoms of vague ill health usually consisting of weakness, dizziness and headache. One case in particular is worthy of note. It involved an automotive repair mechanic who performed his duties in a two-stall garage. During his approximately five years of employment, he periodically complained of headaches, spells of irritability and nervousness. At times, he also found himself stumbling wildly about with the sensation of loss of equilibrium. A number of medical examinations failed to establish anything organically wrong with the patient. The symptoms usually abated after a few days of rest at home only to reappear at various intervals upon returning to work. While he was experiencing one such attack, a sample of blood was obtained and examined for carboxyhemoglobin (COHb). The results indicated a blood level of 18% COHb. A claim was subsequently submitted to the State Industrial Accident Insurance Board.

An investigation of the claimant's working environment indicated that he probably had been

exposed to carbon monoxide since he was primarily responsible for keeping a number of trucks in good running condition. Furthermore, mechanical exhaust ventilation was conspicuous by its absence. Tests with doors closed while the regular tune-up operation was being performed indicated CO concentrations of 300 ppm a few minutes after the engine was started.

A question was raised concerning the possibility of chronic CO poisoning. In the meantime, a questionnaire was sent to a number of other state agencies as well as provinces in Canada regarding this matter.

All of the inquiries were answered in July of 1958, some by medical personnel and others by nonmedical people. Of interest were the answers to the question, "Does your Board recognize chronic CO poisoning?"

State of Ohio, Bureau of Workmen's Compensation: It does not recognize chronic CO poisoning as a clinical entity. Quebec Workmen's Compensation Commission: "We did not have to deal with such class of claims yet." Saskatchewan Workmen's Compensation Board: "Only in one claim did our board recognize chronic CO poisoning, and in this instance compensation was paid in decreasing amounts for a period of approximately three years and three months when payments were stopped with no permanent partial disability being established."

Oregon State Industrial Accident Commission: "Some years ago, largely due to the work of Beck, there was much talk about chronic CO poisoning, especially in relationship to heart disease. It is my impression that most of Beck's conclusions have been disproven. Frankly we look with the greatest doubt upon claims for chronic CO poisoning, usually having the patient examined by an internist discloses some other cause for the complaints listed. In summary we do not recognize chronic CO poisoning."

Michigan Workmen's Compensation Dept.: "On the basis of my experience, and without reference to any particular case, I would have no hesitancy in believing that chronic CO poisoning resulting from the employment would be found to be compensable. By the same token acute CO poisoning would also be compensable." Montana Industrial Accident Board: "We have not seen a case that could be classified as true CO poisoning. We doubt that such a condition would be classified as an industrial accident under the law of this state."

Idaho Industrial Accident Board: "The condition of chronic CO poisoning is recognized." Toronto Workmen's Compensation Board: "We doubt very much if there is such a thing as chronic CO poisoning, or that it has any bearing on coronary heart disease." New York Workmen's Compensation Board: "Our statute covers both accidental injuries and occupational diseases that arise out of and in the course of employment. The courts also recognize repeated trauma as accidental injury where it does not fall within the concept of occupational disease. Awards for acute CO poisoning have been made on the theory of accidental injury. Michelfelder v. Van Alstyne, 217 App. Div. 810, 245, N. Y. 569, relates to bronchitis attributed to an extra amount of CO from a running motor. Richard v. Franklin Mfg. Co. 223 App. Div. 797, involves death due to endocarditis. Although the defendant, an automobile repair man, was exposed to CO over a period of two years as a result of his work in a test shed, the case apparently was considered on the basis of an accidental injury when he suffered a fainting spell. The theory of the case is not entirely beyond dispute, however, since the court majority affirming the award of workmen's compensation death benefits wrote no opinion. In any event, the case rested upon medical opinion that the endocarditis had been aggravated by the exposure to CO gas. In Dagan v. Thorpe, 272 App. Div. 983 where death was due to chronic CO poisoning the award was based upon the occupational disease article of the statute. The cases considered by the courts have involved either death or total disability, but there is no statutory reason for denying benefits for partial disability where substantial medical evidence supports a finding of such disability due to either acute or chronic CO poisoning."

Manitoba Workmen's Compensation Board: "I would think that if chronic CO exposure could be established as arising out of industry, then the claim would be accepted." Alberta Workmen's Compensation Board: "Both acute and chronic CO poisoning are compensable. In 1957, there were 20 claims accepted under both categories."

In Japan, Masanori Hara reports some intriguing work. A health survey[6] concerning hearing, vestibular function and complaints of workers in a factory which had a CO hazard was done. There were a few cases complaining of tinnitus but no cases complaining of dizziness. Audiometric and vestibular function examinations did indicate some impairment in both. Practically all workers with hearing disturbances showed vestibular function disturbances except for a few workers who indicated accelerated functions. No impairment of hearing was noted in workers exposed less than five years.

A survey[7] of 11 males and 48 females was performed by the above mentioned author in a home industry of hemp yarning in which CO was present from a nonvented heating source. This survey consisted of eye and hearing examina-

tions. Workers exposed to CO showed impaired hearing when compared with controls. Those with chronic axial optic neuritis also exhibited hearing impairments and the author concludes that it is conceivable that CO could affect both the optic and auditory organs.

Shiro Kamei concludes in a number of animal experiments that:

1. Urinary thiamine excretion[8] in rats increased with the course of prolonged exposure to CO and that the impairment of phosphorylation of thiamine in chronic carbon monoxide poisoning is suggested.

2. That prolonged and repeated CO inhalation reduced the riboflavin,[9] nicotinic acid, flavinadenine dinucleotide and flavin mononucleotide content of various organs in the rat, and that long-term inhalation of CO may destroy the respiratory enzyme. Furthermore the vitamins necessary for the composition of the respiratory enzymes are also reduced by chronic CO poisoning.

Komatsu *et al.*[10] studied the influence of prolonged and repeated exposure of rabbits to CO on tissue metabolism. They found that hemoglobin content as well as oxygen capacity of the blood was constantly changing and that the oxygen conveying efficiency of the hemoglobin was also variable. The oxygen consumption was decreased in the heart and kidney homogenates, but not changed in the brain and liver. It was, therefore, suggested that the respiratory enzymes of the heart and kidney are remarkably sensitive to anoxia.

A case of chronic CO poisoning verified by a significantly elevated blood carbon monoxide level is reported by Gilbert and Glaser.[11] The patient had anorexia, weight loss, a fluctuating organic mental syndrome and recurrent episode of loss of consciousness and ataxia. After withdrawal from the source of exposure, serial electroencephalograms showed progressive clearing of focal and paroxysmal abnormalities correlated with clinical improvement.

In the *Journal of Applied Physiology*, Wilks, *et al.*[12] report the physiological effects of chronic carbon monoxide exposure. According to the authors' summary, a group of seven dogs was given a daily six to eight hour exposure to CO levels of 0.08 to 0.10% for a period of 36 weeks, and their tolerance to CO was compared with that of normal dogs, altitude-acclimatized dogs, and dogs transfused with blood from normal dogs. The observed increased tolerance to CO was attributed primarily to the increase in concentration of the hemoglobin pigments in the blood. With this increased hemoglobin there was a greater reserve of the pigment for O_2 transport at a given concentration level of COHb. There was no evidence of factors other than hematologic which increased the tolerance of dogs to CO. Animals subjected to other hypoxic stress such as altitude, or normal animals transfused with normal blood to increase the hematocrit level, showed increased tolerance similar to that accomplished by chronic exposure to low CO levels.

Finally, according to an investigation of carbon monoxide concentrations in German coke plants and foundries employing a large work force, it was concluded that chronic CO poisoning does not exist. It was recommended, however, that the expert must decide the question of what CO concentrations in the blood and air in relation to time can lead to subjective and objective symptoms related to CO. So-called chronic CO was replaced by a concept of "mild relapsing CO poisoning." The above was reported at the 13th International Congress on Occupational Health.[13]

This review reflects some of the attitudes that have been encountered. At once it becomes apparent in some instances that what has been termed chronic carbon monoxide poisoning is not other than repeated episodes of acute poisoning. This indicates chronic exposure rather than chronic poisoning. Other situations that have been characterized as chronic poisoning relate to the well-known sequelae that sometimes follow severe acute poisoning. Notwithstanding, there is warrant to iterate the statement contained in the 1930 ILO publication: "The question of the existence or not of chronic CO poisoning has been much discussed and is not yet settled."

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FORWARD STEPS IN MIGRANT HEALTH

Since the early thirties and the days of the "Okies," public conscience has been stirred by the plight of the unfortunate migrants who are both social outcasts and needed workers. Who could lack sympathy when confronted by a mother and her children huddled around a small kerosene stove in a tar-paper shack? And who is not shocked when the local newspaper tells of this same shack going up in flames with all lives lost? For years we have wondered, "What can be done to improve the lot of the migrant worker?"

Health an Important Crop

Of course, we in Delaware are all interested in maintaining the health and welfare of the 7,000 or so seasonal farm workers, for we cannot do without them. The farm economy of Delaware is dependent upon having a large number of seasonal agricultural workers to harvest and process the crops. But, how shall we get to these people with information and let them know of our willingness to help? The problem is complicated by their constant mobility, poverty, lack of education, and lack of community acceptance. Our goal now, as then, is to establish continuity of care by health departments in the so-called "migrant stream," which flows northward from Florida and Georgia through the Carolinas and Virginia into the Delmarva Peninsula and the Middle Atlantic States. If these people have no real "home," perhaps each of the interested states can provide a semblance of one, including the health services to which any local resident is entitled.

Such health services would understandably include immunization, chest x-rays, serological tests and treatment for syphilis, prenatal care and acute medical care facilities. Also there appears to be a real need for some form of health or hospitalization insurance which would not be the usual state-limited plan, but would allow one to contribute a portion of his earnings in each state along the way.

The "key man" in reaching these people is the crew leader, who acts as a father, banker, bill collector, business agent, disciplinarian, etc. for his group. Very likely he will accompany his people all the way from Florida to New York and back again. It is obvious, then, that we must deal with such men, no matter how unscrupulous some of them may be. An added reason, in the case of the Puerto Rican workers (10% of the total), is the ability of the crew leader to translate our plans from English into Spanish. (Some of us have subsequently studied Spanish, so we are no longer at his mercy).

Extension of Health Care

Besides the regularly scheduled clinics and conferences of the county health departments, which have always been open to migrants and their families, we feel that some of our services must be carried to the camps. One example, since 1957, has been the Well Child Conference at Staytonsville, where the Delaware Council of Churches (Migrant Ministry) maintains a Day Care Center for children of migrant workers. Here the usual type of pediatric clinic may be seen, where the clinician discusses growth and development of the child and gives the necessary immunizations.

Gaps in our program which eventually must be filled are: (1) the expansion of our prenatal clinics from the two now existing in Sussex County (quite often a migrant's wife receives her first ante partum attention after she arrives here); (2) the development of hospital outpatient facilities in the two lower counties.

Added Services

Another service rendered annually for the past three years concerns the control of tuberculosis and venereal disease. This program is one of those fine, heart-warming examples of cooperation among official and voluntary agencies, in this case, the Delaware State Board of Health, the Delaware Anti-Tuberculosis Society, and the

Venereal Disease Division of the U. S. Public Health Service. Since budget and personnel are shared, the cost of the program is not especially burdensome to any one agency.

A health educator assigned specifically to this project contacts each of the camp operators and does the scheduling approximately two weeks in advance. The value of one man being responsible for this work is shown in the lack of confusion and duplication, the scheduling of workers from several farms at one unit location on a certain day, and the ability of the Mobile Unit to pick up several locations on one trip. The day before a farm is to be visited, this same health educator calls again to make sure all is in readiness.

Mobile Unit Operation

The Mobile Unit carries with it a full complement of two x-ray technicians, two blood-letters and two clerks. As each laborer walks on the bus, the registration blanks are filled out, one for chest x-ray and one for serology. Social Security cards, if available, are used for individual identification. Let us see how this program succeeded in 1958.

The 70 mm. films were developed and read within three days. Of the total 4,117 x-rayed, 39 or 0.9% were recalled as Tuberculosis Suspects. Thirty-five of these came into the county health unit for a 14"x17" film and a more complete history after the employer had been contacted by the Public Health Nurse. To give credit where such is due, we must note here that some of these "retake" clinics were held in the evening, and that our personnel willingly worked long hours for the success of this venture.

Of these 35 persons who actually showed up for a large film, 10 were categorized as "Urgent"; eight fell into the active classification, one never had a diagnosis established, and the other one left Delaware before diagnosis could be completed. Thus we may conclude that approximately 0.2% of our particular sample of migrants had active tuberculosis, certainly a much higher yield than that obtained from the local community-wide survey.

Results of Serology Survey

Blood specimens were obtained in a Sheppard vacuum tube which is a sterile, self-contained unit. The VDRL test was performed on each specimen in the State Board of Health laboratory, and the result known within 24 hours. Speedy work and good interdivisional cooperation meant that the reactor could be checked and, if necessary, treated before he moved on to the next state.

Of the 4,039 migrants' blood tested, 488 or 12% were found to have a positive VDRL. Each reactor was checked through the central registry to see if we had treated him previously for syphilis. If not, an epidemiological form was made out and assigned to a VD investigator, with the hope that this individual could be found and brought in to a VD clinic in the county health unit. At this clinic, a complete history was obtained and written on his record by the examining physician.

Now, it used to be a common saying among those who worked with VD in migrants that "they may not be the *best* treated people in the country, but they are certainly the *most* treated!" We have tried to eliminate this condition by treating for syphilis only those migrants who showed a high titre, gave a history of previous inadequate treatment, or had no treatment at all. Eventually we hope that more extensive use of the "health card" by all the states in the migrant stream will decrease the need for even some of this treatment. Undoubtedly there are still some migrants who are subjected to an injection of 2.4 million units of benzathine penicillin G (Bicillin) every time they enter another state with a positive serology. In our case, 70.2% of these reactors in 1958 were felt to require treatment.

The Clinic Comes to the Farm

There were times last year when we realized that the farmer-employer might resent our taking away his workers to the clinic for diagnosis and treatment. The alternative was to bring the clinic to the farm. A physician and a VD investigator visited the farm and held clinic in any appropriate place. . . . sometimes in a tool shed, a bunk house, or even the back seat of the automobile. We are sure that the farmer appreciated our efforts to keep his men on the job.

The 4,000 or more migrants who thus went "through the mill" in 1958 received *health cards* within a week from the time they stepped off the Mobile Unit. We know, from conferences held with our counterparts in other states, that the Delaware health card is now recognized and honored. This little wallet-sized card gives the following information: name and address, state or country of origin, social security number, work number (given by the Farm Placement Unit), date and results of chest x-ray and serological test for syphilis. The individual worker leaves Delaware with the feeling that here we are enough concerned to do all in our power to promote his health and well-being.

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U. S. Department of Health, Education, and Welfare
Public Health Service

A NEW DIVISION OF OCCUPATIONAL HEALTH

A major focus of the current reorganization to enable the Public Health Service to cope more effectively with the changing national health scene is the strengthening of environmental health activities. The increasing importance of the occupational health segment was recognized in the recent creation of a Division of Occupational Health. Dr. Harold J. Magnuson, career officer, who has directed PHS occupational health activities for the past four years, is in charge of the new Division. Replacing the former Occupational Health Program, the new Division will be able to provide long-needed increased effort in this health field, as recommended to the Surgeon General by the Study Group on Mission and Organization of the Public Health Service.

The Division will direct, coordinate and develop the Public Health Service's nation-wide program to protect and improve the health of American workers — with particular emphasis on the prevention of diseases of occupational origin.

To keep pace with rapidly changing technology and multiplying hazards in the work environment, the Division will carry out an expanded research program to yield better techniques, materials, and equipment for use in the prevention, diagnosis, and treatment of occupational disease. Research efforts will be intensified in toxicology and in related fields of clinical medicine, engineering, chemistry, and physics. Also, the physiologic and psychologic factors in the work environment will come under more penetrating study.

Greater emphasis will be placed on the training of personnel in occupational medicine and nursing and in industrial hygiene, to meet the needs of this rapidly growing field. The Division will work with academic institutions and other groups to help develop the necessary professional resources.

A third major expansion will be in state aid services, designed to support and assist official agencies in the development and operation of occupational health programs throughout the nation.

Stimulating effective community and industrial efforts to supply occupational health services will be stepped up.

The Division of Occupational Health will function through headquarters in Washington, the Occupational Health Research and Training Facility in Cincinnati, and the field station in Salt Lake City.

Nature and Scope of the Problem

The occupational health problem is described by Dr. Magnuson as two-pronged, involving (1) the identification and study of occupational diseases, and (2) occupational disease control and worker health maintenance through optimum utilization of industrial hygiene and preventive medical services.

The problem is compounded by the lack of adequate reporting of occupational diseases. Of the 50 states, 28 have laws requiring medical reporting of occupational diseases, but only three states receive 100 or more reports per year.

Based on reports of governmental agencies with the most comprehensive statistics, an extrapolation suggests a national incidence of 200,000 to 400,000 cases of occupational disease annually. Dr. Magnuson believes that even the 400,000 figure may be conservative because the reports are based on the most easily recognized occupational diseases, and do not reflect those occupation-related diseases that masquerade under diagnoses of other illnesses. The belief is growing that there is a large gray area of such diseases, that cumulating knowledge and further research will define them more sharply.

The difficulty of achieving control of occupational diseases seems staggering, when one reckons with the size of American industry. Though a popular image of industry is "big," small plants with less than 500 workers represent over 99% of all establishments, and employ 60% of the nation's nonagricultural workers. The small size of these plants presents both economic and manpower resource problems, and these two elements largely account for the lack of basic industrial hygiene control measures in over half the industrial plants in this country.

Dr. Magnuson expects the manpower shortage in occupational health to worsen. According to present growth rates, manpower estimates for 1970 are: industrial physicians 2,900; industrial nurses 26,000; and industrial hygienists 1,600. These figures fall far short of the estimated need: 4,800 industrial physicians; 35,000 industrial nurses; and 3,400 industrial hygienists.

The hazards to which workers are exposed are constantly multiplying from a prolific technology. The sharp rise in the production index of chemicals and allied products from 97 in 1947 to over 200 in 1959 is illustrative of technologic growth. Of the 10,000 to 20,000 chemicals developed each year, estimate is that 500 find commercial application. Although many of these are potentially toxic, the high cost of toxicologic testing, estimated as $100,000 per compound, precludes adequate evaluation. Furthermore, Dr. Magnuson observes, as in any other medical problem, animal testing is but the beginning and the final answer requires prolonged, careful observation of exposed humans.

The forces shaping occupational health, according to Dr. Magnuson, include such diverse influences as legal compensation, humanitarian factors, scientific and technical advances, patterns of medical care, absenteeism and medical care costs to labor and management, and availability of trained manpower.

Since, in large plants, the forces shaping the pattern of occupational health differ from those in small establishments, methods of achieving the common goal of worker health must essentially differ. A common pitfall cited by Dr. Magnuson has been the failure to distinguish between those problems which must be met in the in-plant environment (control of occupational health hazards) versus those problems that may be met in the in-plant environment or elsewhere (general preventive health maintenance).

Long-Range Plans

To meet the basic occupational health problems, the Division has set 10 long-range goals:

Achievement of adequate reporting of occupational diseases.

Recognition of occupational factors contributing to the etiology and progress of diseases.

Extension of environmental controls to workers in all plants, large and small.

Development of more efficient methods of bringing preventive medical services to workers, particularly in small business.

Establishment of effective occupational health programs in every state.

Improvement and development of curricula to include pertinent segments of occupational medicine and industrial hygiene, for the training of students in business administration, medicine, nursing, and engineering.

Achievement of increased knowledge among health practitioners regarding diagnosis, treatment, and prevention of occupational diseases.

Achievement of increased awareness and knowledge, by workers and the general public, in how to prevent occupational diseases.

Development of international exchange of occupational health knowledge, and methods for its application.

Development of physical and human resources adequate to accomplish the above goals.

Aims and Activities

Directed toward the goals just mentioned, the aims of the Division are: (1) to develop better techniques, materials, and equipment for use in prevention, diagnosis, and treatment; (2) to stimulate effective community and industrial efforts to supply occupational health services; (3) to assist Federal, state, and local agencies, health practitioners, labor unions, management, and individual workers (a) in the application of knowledge required to prevent occupational diseases, and (b) in the development of optimum ways of bringing preventive services to the working population; and (4) to determine the health status of the American labor force and the total impact of the working environment upon human health.

Activities will be carried out by laboratory and field studies; demonstration, consultation and educational activities; training and the production of training aids; study of social and economic problems involved, continuous evaluation of all available data; grant mechanisms; and joint undertakings with other parts of the Bureau, the Service, and other resources of the nation.

The responsibilities for these activities will be carried out through the:

Office of the Division Chief Dr. Magnuson: *Acting Executive Officer* Frank L. Worden; *Program Planning and Analysis Chief* Dr. W. Clark Cooper; *Information Officer* Mrs. Tula S. Brocard.

State Services Branch (Washington): *Chief* Henry N. Doyle; *Employee Health Programs Section Chief* Dr. Thomas K. Callister; *Occupational Nursing Section Chief* Mrs. Mabelle J. Markee.

This Branch participates in developing and strengthening occupational health programs in state and local governmental agencies; in providing consultative and other technical services to states, through states to employers and labor groups, and to foreign countries; in promoting preventive health services for places of employ-

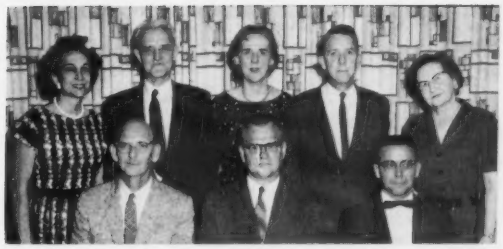

Figure 1
Division of Occupational Health
Washington Professional Staff
Left to Right, Row 1: Henry N. Doyle, Chief, State Services Br.; Dr. Harold J. Magnuson, Division Chief; Frank L. Worden, Acting Executive Officer. Row 2: Mrs. Tula S. Brocard, Information Officer; Dr. William M. Gafafer, Technical Advisor; Mrs. Eloise Worden, Public Health Advisor; Dr. W. Clark Cooper, Chief, Program Planning and Analysis; Mrs. Mabelle J. Markee, Chief, Occupational Nursing Section. Not present for picture: Dr. Thomas K. Callister, Chief, Employee Health Programs Section; Margaret F. McKiever, Research Analyst.

ment, and in maintaining up-to-date information on the status and trends of health services and health insurance programs in places of employment and in labor groups; in developing occupational health nursing services; and in providing occupational health nursing consultation to states and through them to industry, universities, and labor groups. The Branch contributes to a broad program in occupational health research and training.

Research and Technical Services Branch (Cincinnati): *Chief* Dr. Douglas H. K. Lee; *Toxicology Section Chief* Herbert E. Stokinger; *Dermatology Section Chief and Acting Occupational Medicine Section Chief* Dr. Donald J. Birmingham; *Physical and Chemical Analysis Section Chief* Robert G. Keenan; *Engineering Section Chief* Robert L. Harris.

This Branch is responsible for laboratory and field research and study in clinical medicine, toxicology, environmental engineering, human biology, and ecology for the improvement, recognition, understanding, and control of occupational health problems; development of clinical and engineering methods for the control of occupational disease; providing scientific and technical advice in the areas of its responsibilities, as needed by the Division or by other official agencies, unions, industry, and academic institutions; and for reporting the research achievements of the Branch through appropriate technical publications to the professional community at large.

Training Branch (Cincinnati): *Acting Chief* Lewis J. Cralley; *Requirements Analysis Section Chief* Charles D. Yaffe; *Training Operations Section Chief* Andrew Hosey; *Technical Information Section Chief* Dohrman H. Byers.

The Branch analyzes and evaluates the occupational health training needs of Federal, state, and local governmental agencies, unions, and industry; formulates training programs, and cooperates with academic institutions to meet the training needs; arranges for training courses to be conducted by personnel drawn from other Divisions, academic institutions, other sources, or from consultants; coordinates training activities of the Division with other Service training programs; prepares training aids; maintains for public use a technical information service on occupational health hazards, toxic substances used in industrial processes, detection methods, and general control procedures; and cooperates in international exchange of technical literature and abstracts.

Salt Lake City Field Station is under the direction of Duncan A. Holaday. This station provides for a wide range of Division services to the western states.

Some examples of progress in occupational health activities are:

Study of Silicosis: Over 10,000 employees in 38 different types and sizes of metal mines in 12 western states and Missouri have participated in the medical-environmental study being conducted by the Public Health Service in cooperation with the U. S. Bureau of Mines. This marks

Figure 2
Division of Occupational Health
Cincinnati Executive Staff
Left to Right, Row 1: Dr. Herbert E. Stokinger, Chief, Toxicology Section; Dr. Lewis J. Cralley, Asst. Chief, Research & Technical Services Branch and Acting Chief, Training Branch; Dr. Douglas H. K. Lee, Chief, Research & Technical Services Branch; Charles D. Yaffe, Chief, Requirements Analysis Section, Training Branch; Dohrman H. Byers, Chief, Technical Information Section. Row 2: A. M. Doyle, Administrative Officer; Dr. Robert H. Flinn, Asst. Chief, Occupational Medicine Section; Robert G. Keenan, Chief, Physical & Chemical Analysis Section; Andrew Hosey, Chief, Training Operations Section; Robert L. Harris, Chief, Engineering Section. Not present for picture: Dr. Donald J. Birmingham, Acting Chief, Occupational Medicine Section and Chief, Dermatology Section.

the completion of the Western phase of this investigation, and the study teams are now planning to move into the eastern states.

Uranium Miners: Studies of uranium mines in the Colorado Plateau have continued in collaboration with the National Cancer Institute, the AEC, and the states. The Division of Occupational Health coordinates environmental sampling and cooperates in medical surveillance. Two thirds of the mines surveyed had concentrations of radioactive dust and gas exceeding recommended working levels. Annual census of all miners, year-round follow-up, and triennial examination show lung cancer deaths are in excess of predicted numbers, but difference is not statistically significant in view of small number of miners who have had long exposures. The total number of uranium miners has now passed 5,000.

Noise: A study of the effects of noise in Federal Prison Industries has been completed. Preliminary analysis of the data indicates that noise exposures of about 90 decibels, not unusual in industry, will cause some loss of hearing and that recovery is not complete in many workers exposed for over six months.

Development of Analytic and Diagnostic Methods: Ion exchange methods have been developed for rapid quantitation of fluorides in urine which will greatly facilitate the study and control of occupational hazards in the superphosphate, aluminum, and steel industries. A micromethod for estimating blood level is being evaluated.

Emergency Services: Emergency service was given following reports of injuries from Haitian voodoo dolls. The presence of allergenic and toxic substances was demonstrated by study at our field headquarters. In cooperation with state and local health authorities, dolls were removed from the market and their importation halted. An outbreak of severe asthma, rhinitis, and gastrointestinal symptoms in three eastern seaports among workers handling shipments of castor bean pomace from Brazil, and attributed to failure on the part of the processor to steam-treat the product, was studied in 1959. Emergency control measures were recommended and the importer agreed voluntarily to accept no further shipments pending assurance as to reduction of the hazard.

Training: Sixteen training courses and 12 seminars were held in the fields of industrial hygiene and chemistry, control of uranium mining and milling hazards, industrial noise control, and industrial nursing. These were attended by 1400 persons from state and local health departments, universities, the armed services, and industry.

Consultative Services to State and Local Health Departments: The Division of Occupational Health, in cooperation with the Kansas State Health Department, investigated complaints of lead poisoning in a lead solder plant. Fifty-seven workers were examined and the findings reveal a definite lead hazard. An environmental investigation will follow.

Cooperative Studies: The Division of Occupational Health, in cooperation with the Michigan State Health Department, in 1959 and 1960 conducted a study of the cause and prevention of celery blisters in the Michigan celery growing areas. The cause of the celery blisters was shown to be a phototoxic material in "pink rot," a fungus disease of celery. Effective preventive measures were recommended.

A study was made of the medical records of beneficiaries of the International Longshoremens' and Warehousemen's Union-Pacific Maritime Association Welfare Fund in Stockton, California, done under contract with the San Joaquin County Medical Society. Objective was to obtain morbidity data and to evaluate applicability of such records to obtain statistics on disease as related to occupation.

The Division of Occupational Health is cooperating with the US Navy in nuclear submarine habitability studies. Atmospheric gas concentrations and particulates have been measured and air ion content determined.

Moving ahead in the field of occupational health is urgent, Dr. Magnuson emphasizes. In the words of Surgeon General Burney, "The nation's population and industrial expansion give us the picture of a strong and growing economy. A key factor in that growth is the health of the American people."

Our population is, indeed, much smaller than those of the two communist powers, Russia and China. Yet our people have had the health and vigor to develop a technology that is a marvel to the world, to achieve an astonishingly high standard of living, and to provide manpower for national defense. It is not the labor-saving devices alone that increase our capacity to produce, but also the quality of our productive manpower.

We anticipate a growing population in the United States. But the world population is increasing, too; and in many areas, from a much larger base than ours. If our country expects to continue expanding the economy, therefore, we must do better than maintain our present high level of health. We must, instead, press for a rising health standard.

THE WET DRESSING

Its Use in Acute Cutaneous Inflammation

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Despite recent advances in systemic therapy, the topical application of various medicaments is still the preferred treatment in most of the dermatoses affecting the glabrous skin. In these days of "magic" lotions and salves, the practitioner should always be mindful of the adage which admonishes him "first, do no harm." It is advisable to initiate topical therapy with a bland remedy, soothing, but not necessarily inert. Almost without exception his efforts will be rewarded by subjective and objective improvement and a pleased patient. It is a sad reflection on the teaching of clinical dermatology to find so many practitioners unacquainted with the basic principles of topical dermatologic therapy.

Duhring noted that the "successful treatment of skin diseases depended upon a thorough knowledge of the pharmacologic action of the remedy employed." The cool wet dressing correctly applied remains one of the most important modalities. Nothing affords such prompt and pleasing relief to the inflamed skin. Many effective therapeutic procedures are possible with water, in which various substances have been dissolved or suspended. The industrial physician can readily appreciate the benefits to be derived from the wet compress treatment of acute contact dermatitis due to noxious chemicals.

Wet compresses are indicated in any acute or subacute inflammatory process — contact dermatitis, allergic eczema, plant dermatitis, anogenital pruritus, acutely inflamed ulcers, erysipelas, insect bites, and lymphangitis. As a preparatory medication in hypostatic ulcer and hypostatic dermatitis prior to application of a glycogelatin (Unna's) boot, such as Dome-Paste* Bandage, they are effective. Similarly, in acute dermatophytosis of the hands and feet, pompholyx and dyshidrosis, they are also indicated.

Wet compresses are of great value in branches of medicine other than dermatology and general surgery. Burow's solution compresses are indicated in all acute inflammatory diseases of the eyes and eyelids. In fact, von Burow was an ophthalmic surgeon and suggested the Burow's solution which we use in a modified form today. Another use is in otology. In recurrent furunculosis of the external auditory canal, wicks saturated with Burow's solution and inserted into the canal afford prompt relief. The use of the Domeboro* tablet or packet for Burow's solution is convenient. Other diseases of the external auditory canal and auricle respond promptly with relief from itching and burning. Relief is also obtained in seborrheic dermatitis, contact dermatitis and neurodermatitis in an acute flare. Seborrheic dermatitis, psoriasis and streptococcal intertrigo, when occurring around the external ear, frequently cause painful and pruritic fissures. Narrow strips of soft muslin saturated with Burow's solution and carefully placed over the fissures are quite effective in relieving the acute edema and discomfort.

In gynecology, for acute vulvitis and pruritus vulvae, cold compresses of Burow's solution, or Soyboro* solution, afford relief, especially when skim milk is used as the vehicle. As a vaginal douche, Domeboro* solutions promote the normal acid pH of the vagina and are helpful in suppressing Trichomonal infections. Hot compresses afford relief from pain and hasten the course and resolution of acute Bartholinitis.

The practitioner must be familiar with the indications and select the proper type of wet dressing and correct method of application. He must then decide on the active medication to be incorporated in the dressing. An appropriate chemical improperly applied may do harm.

Wet dressings are customarily of three types: compresses, poultices and moist chambers. These dressings possess varied physical and chemical properties which may be subdivided as follows:

I. Physical Action
   A. Detergent Effect
   B. Emollient Action and Heat Dispersion
   C. Heat Retention
II. Chemical Action
   A. Antisepsis
   B. Keratoplasia
   C. Buffer Action

During the past decade many additives for

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*Reg. T. M. Dome Chemicals, Inc., New York, New York.

the wet dressing have been developed, affording better formulation control and greater efficacy with consequent superior benefit.

I. Physical Action
A. Detergent Effects

Darier has said that "no one would dream of interposing a sheet of paper or the patient's shirt tail between an application and the diseased skin." However, the practitioner does the equivalent when he neglects to cleanse a lesion prior to medication. Crusts, scales, hairs, previous medication, secondary products or foreign material must be removed before the active, curative medication can reach the living diseased cells. Otherwise, the medication is inert. It is often necessary to cleanse a lesion in order to make a diagnosis. For this purpose, water is most frequently used either alone or emulsified with some oily substance.

For the removal of crusts and ordinary loose scales from the skin, nothing is superior to Burow's solution (Aluminum Acetate Solution, USP). A convenient, modernized Burow's solution may be prepared by dissolving one Domeboro* tablet or Domeboro* powder packet in a pint of water. Several thicknesses of soft, washed, finely woven material as an old sheet or pillowcase are saturated with the solution and applied to the lesion for several hours. The dressing should be completely changed every half hour, but in the interim, the solution should be frequently replenished, sufficient to keep the dressing sopping wet. It may be used hot or cold. When applying wet compresses to the fingers and toes, one should insert a double layer of material between each digit. For adequate action it is seldom necessary to use a compress for over 24 hours.

Tap water alone, boric acid and isotonic (or slightly hypertonic) saline solution are also used.

Urea is a cheap and useful detergent. Two or three tablespoonfuls of the crystals when dissolved in a quart of water and applied on soft muslin or stockinette will digest crusts and loosen scales.

A seldom used but effective detergent is the starch poultice. This is prepared as follows: one teaspoonful of crystalline boric acid and four tablespoonfuls of ordinary laundry starch are thoroughly mixed and a paste made by the addition of a small amount of cold water. Then a pint of boiling water is added and the mixture stirred in a receptacle on the stove until a clear, thin, gelatinous solution develops. This indicates bursting of the starch cells. When cool, cut into slices a half inch thick and apply to the lesions between two layers of light, coarsely meshed gauze. This dressing should be changed every hour. Do not let it dry. The starch poultice should be prepared fresh daily and be kept in the refrigerator.

If the lesion is covered solely with scale as in seborrheic dermatitis or psoriasis of the scalp, an oil-in-water emulsion may be prepared as follows:

1.	Domeboro*	½ tablet
	Water	250 cc
2.	Cottonseed oil	100 cc
	Triton - X 45	10 cc

Dissolve the ingredients of each and then mix 1 and 2. The scalp is saturated with the mixture and a handkerchief, knotted in the four corners, is applied to the head. After several hours any ordinary shampoo may be used to remove it. Do not use a fine comb or any other means other than the shampoo to encourage removal of the scales. It may be necessary to repeat this procedure several times. This emulsion may be used also to clean off old medication, grease, crusts and scales from lesions of the glabrous skin.

Olive oil is also a convenient, mild, relatively odorless product used for softening and removing crusts. The addition of a maximum of 1 or 2% salicylic acid increases the keratolytic and detergent actions of the oil. On the scalp it may be applied by the handkerchief method. If for some particular reason more salicylic acid is desired for increased keratolysis, castor oil may be substituted for part of the olive oil due to the greater solubility of salicylic acid in the castor oil.

Dissolve:

Salicylic acid	5
Castor Oil	50
Olive Oil	50

Castor oil is not used alone since its consistency varies too much with changes of temperature.

Still another detergent of this type, recommended by Klauder is:

Sulfated olive oil	55
Light liquid petrolatum	45
Gelatin (25% aqueous solution)	10

This is mixed with two or three volumes of water and applied as a wet dressing.

B. Emollient Action and Heat Dispersion

The dispelling of heat or cooling of a lesion constitutes the most common use for wet dressings. The therapy is usually designated as "wet compress" or "open wet dressing." Best results are achieved by avoiding both ponderous dressings and anything else that occludes or prevents evaporation. Emollience and heat dispersion lend themselves to joint discussion since the dispersion of heat in a lesion has a soothing or emollient effect.

The emollient and heat dispersing activity of

the cool wet compress is a consequence of several factors:

1. Dispersion of heat, both by radiation and as a consequence of evaporation of the vehicle. (Evaporation is a cooling process.)

2. Vasoconstriction with consequent acceleration of the capillary and arteriolar circulation in the inflamed area.

3. Encouragement of drainage. This is especially true when using hypertonic solutions.

4. Consequent relief of the inflammatory process, exudation of lymph, dispersion of readily mobile inflammatory infiltrates and relief of subjective symptoms (itching and burning).

The cooling wet compress functions as well as a detergent in the removal of crusts, scales and detritus. It is essential that it be kept sopping wet because if it dries it can be quite irritating. Ordinary tap water may be used although it is advisable to boil and cool it before use.

Evaporation naturally results in an increased concentration of active ingredients. Replenishment with plain water may be advisable. The dressing material should be as in detergent dressings. Never use absorbent cotton anywhere in the dressing and never use gauze next to the skin. Never cover the dressing with rubber dam, oiled silk, cellophane or any material which would interfere with evaporation. The dressing should be changed or rewetted every five minutes, new pads of material being soaked in a bowl of solution at room temperature; or the bowl of solution may be placed in another larger container holding cracked ice. Pouring fresh solution under one edge or corner of the dressing and expecting it to saturate the entire dressing is a forlorn hope. Since the compress is not bandaged, the entire dressing can be removed and a freshly wetted one applied. Do not put an icebag over a wet dressing to cool it as there is danger of freezing and also of chilling the patient. It is seldom necessary to use a wet compress over two or three days. After permanent removal, because of dryness of the skin, a bland oil or ointment should be applied.

In the elderly and infirm patient avoid wet compresses on extensive areas. It is best not to use them at all on the chest, arms or neck (danger of pneumonia). Likewise, many individuals cannot tolerate cold aqueous compresses on the scalp. If compresses are not continuously applied (and they seldom are), or if it is desired that they be discontinued during the night or periods of rest, apply a bland oil to the area. Wet compresses have a natural tendency to dry the skin.

The following comments deal with the most frequently used solutions and suspensions:

1. *Clear tap water, preferably sterile but not always necessary.* It may be irritating on the eroded skin if the water is hard. If wet compresses of water are continued for too long a time, they may cause hydrops or "waterlogging" of the skin due to hypotonicity; this favors monilial infection.

2. *Skim Milk.* Milk from which the cream has been removed is approximately an isotonic solution. Besides proteins, it contains various salts, especially phosphates of calcium, potassium and sodium, is amphoteric and makes an excellent buffer. It is superior to water as a vehicle for most purposes, especially as an antipruritic. Skim milk may be readily prepared by mixing 1⅓ cups of powdered dry milk in a quart of water.

3. *Physiological Saline Solution* (0.9%). Particular care should be observed in rewetting this dressing because sodium chloride diffuses readily and there is also a tendency to hypertonicity with excessive escape of lymph and serum. Physiological saline solutions may be made extemporaneously by dissolving one level teaspoonful (about 4 gm.) of sodium chloride or bag salt in a pint (about 500 cc.) of boiled tap water.

4. *Aluminum Acetate Solution, U.S.P.* (Burow's Solution). A convenient Burow's solution may be prepared by dissolving one Domeboro* Tablet or one envelope of Domeboro* powder in a pint of water, or a pint of skim milk. A convenient concentrated Burow's solution (Domeboro* Concentrated) is available.

5. *Magnesium Sulfate (Epsom salts) Solution.* One half to one ounce in a quart of water. This is approximately 3%. Epsom salt solutions are used only when one desires the benefits of hypertonicity. It does not diffuse readily and is superior to other salts in attracting water to itself. Although I have indicated its use in 3% concentrations, it has been used as high as 25% in the treatment of superficial burns. It was formerly extensively employed by subarachnoid injection for the production of surgical anesthesia but has no anesthetic action whatever on either the eroded or intact skin.

6. *Sodium Sulfate Solution.* Although not, per se, an antiseptic, this is used for that purpose in the treatment of open wounds and nonmalignant ulcers. Saturated solutions are used (about 65%). It is applied by the same method as any wet compress, so I have mentioned it here. It may also be applied in a wet chamber which is discussed later. Because of its extreme hypertonicity it causes a profuse serous and lymphatic exudation, simultaneously attracting leukocytes and reducing inflammatory edema. Naturally the dressings must be renewed frequently. It is used at tepid temperatures.

7. *Colloid Powder.* Demulcent and emollient

wet dressings containing starch products have been used for years. Corn, potato, linseed, oat, orris root, rice, soybean, and almond meal starches have been used. In my experience, soybean has met with most sustained success.

In the treatment of anogenital pruritus, perianal dermatitis and inflamed hemorrhoids, Marshall, a proctologist, recommends the following procedure as superior to the sitz bath. A turkish towel is draped over the toilet seat (with lid down) and a strip of soft muslin folded to approximately three inches in width by a foot in length, and previously soaked in Soyboro* solution or Soyaloid* solution is spread over the center of the towel. The patient then sits with the buttocks on either side of the strip. This assures excellent and complete contact of the dressing with the entire anal area, which is conveniently stretched, exposing all furrows and fissures to the medication.

8. *Colloid Regional Bath.* One type of therapy which has been neglected in this country in recent years is the "regional bath" — the immersion of the diseased part or area into a medicated bath. This does not differ so much from the wet dressing, except that exposure is for a shorter period and the "regional bath" is preferably warm. Its actions may be summarized as follows:

1. Cleansing effect, 2. Softening of the horny layer, 3. Stimulation of circulation, 4. Tonic effect on the nervous system (especially hot foot baths,) 5. Antipruritic effect, 6. Decongestive (antiphlogistic effect) benefit.

Most regional colloid baths formulations of past years have been either too alkaline or too drying. Others leave a slippery film on the tub. Others clog drains. Sodium bicarbonate, sodium perborate, sodium hexametaphosphate, boric acid and other crystalline salts were used. A preparation which takes advantage of the buffering, antiphlogistic and demulcent properties of the soybean is Soyaloid* Colloid Bath Powder. It provides a convenient, colloid-bath treatment for allergic dermatitis, contact dermatitis, and especially detergent dermatitis of the hands in housewives and industrial workers. I have never been too enthusiastic over the substitution of the synthetic detergents for a good soap. Many women can continue to use soaps if subsequently they just dip their hands into a solution of Soyaloid* Colloid Bath or rub a teaspoonful of it into their hands. It also makes an excellent solution for bathing tired or inflamed feet. In a sitz bath in anogenital pruritus, inflamed hemorrhoids, anal fissures, etc., it also is acceptable.

9. *Rice Water.* This is a popular solution for wet dressings in the Orient although used here in recent years. It requires some time to prepare. Two ounces of clean rice are added to two quarts of water. This mixture is boiled for an hour and a half and then strained. The filtrate has a slightly acid pH and serves well as a demulcent and emollient wet compress. It should be refrigerated if kept for a long time.

10. *Chamomile.* Chamomile flowers, elderflower blossoms, lime blossoms, primrose petals, dog fennel and other flowers have been used as decoctions and infusions for wet dressings for many years, especially in Europe. I recall a young British miss whom I attended many summers ago for a particularly severe attack of prickly heat. She insisted that nothing would help her other than cold compresses of chamomile tea which she had used in England on previous occasions. At that time it was a major project to obtain the powdered flowers in New York; but I succeeded and the results were miraculous. Now powdered matricaria (German chamomile) packets as Chamo* Powder, each containing the extractives of two grams of the flowers are available. Chamomile makes a soothing, antiphlogistic wet compress for the inflamed skin. The content of one packet is dissolved in a pint of water.

Matricaria, N. F., is the official designation for the dried flowerheads of Matricaria Chamomilla. It contains a volatile oil consisting of azulene, sesquiterpenes, sesquiterpene furfurol and a bitter substance, possibly a fatty acid. Azulene is presumably the principal antiphlogistic agent and largely responisble for the relief obtained following the use of chamomile wet compresses.

C. Heat Retention

We desire to retain heat in the tissues for many localized infections, cellulitis, abscess, furuncles, carbuncles and felons. In lymphangitis and erysipelas cold compresses are preferable. Hot wet dressings have largely replaced the old fashioned poultices and cataplasms, and are certainly more sanitary when we wish to apply moist heat to the skin.

Almost any of the solutions recommended for heat dispersion may be used for this purpose, too, but it should be appreciated that we wish now to protect the tissues from loss of heat. Gauze may be used with absorbent cotton interposed between the layers, more or less as an insulator. Do not apply the cotton directly to the skin. The entire mass is saturated with the desired medication and a bandage or stockinette applied. Cellophane, rubber dam or other impermeable material may be used outside the dressing beneath the bandage to retard evaporation and drying. Pliofilm mittens for the hands and socks for the feet are commercially available or can be easily made. It is not necessary to keep a wet poultice sopping wet. It may be moistened by occasional

immersion or injection of solution under the bandages. Wet dressings in which the medicament is a dissolved crystalline salt (i.e., Burow's solution, saline, boric acid) cannot be satisfactorily rewetted if they have accidentally dried completely. A fresh dressing should be applied. When extensive areas are covered it might be advisable to insert fenestrated rubber or plastic tubing through which more solution may be injected by syringe or constant drip.

II. Chemical Action
A. Antisepsis

While it is true that many of the ostensibly bland emollient solutions exert some antisepsis, more active germicides may be employed. It must be appreciated that antiseptics are often injurious to tissue cells. It is therefore, with few exceptions, preferable to use one that is mild, one which will aid the normal immunologic and cytophylactic properties of the skin. That is one reason why wet dressings such as Burow's solution,* Soyboro solution and Chamo* Powder solution are usually to be preferred, even in infected lesions. Over half of the patients coming into the dermatologist's office with infected cutaneous lesions, bacterial and fungal, are victims of too vigorous topical therapy with bactericidal and fungicidal medications. Paradoxically, in most all, the induced treatment dermatitis and the original infection both disappear with the mild soothing wet compresses.

Of the common antiseptics used as wet dressings the following are examples, with pertinent comment.

1. *Boric Acid (1-2%).* This should be avoided on the eroded skin and over large areas, especially in children where boron poisoning is an actual hazard. Boric acid is only moderately soluble in water at room temperature so that there is also danger of crystallization in the dressing, with consequent irritation.

2. *Bichloride of Mercury (1-5000).* This is preferably applied as hot as can be borne by the patient. Avoid its use on extensive areas. The earliest symptom of toxic absorption is salivation. Bichloride of mercury and saturated solutions of sodium sulfate lend themselves admirably to use in deep, penetrating ulcers and infected sinuses when applied as a closed moist chamber dressing. The lesion is first thoroughly cleansed and cleared of detritus and necrotic tissue; then packed with wicks, thoroughly wetted with the solution until full. The entire area is then covered with several thicknesses of gauze, also wetted. Over this is spread a layer of vaseline gauze, extending beyond the lesion. This in turn is covered by rubber dam or pliofilm, the free edges secured to the skin by plastic adhesive plaster or liquid latex. The dressing will remain moist for a long time, but it is advisable to redress completely every 12 hours.

3. *Potassium Permanganate Solution (1:4000 to 1:10,000 freshly prepared).* This is one of the most overrated and most unsatisfactory of all wet dressings. It is a poor antiseptic, a mediocre oxidizing agent, although it is a satisfactory deodorant. The Federal Food and Drug Administration has recently issued warnings on its promiscuous use. When allowed to dry on the skin, which can hardly be avoided, it causes a dirty stain; it may cause burns, and almost invariably in moist lesions an occlusive tough crust, favoring retention of purulent exudates. Severe flares with lymphangitis and erysipelatoid eruptions often follow its use.

4. *Dalibour Water.* There are few applications for dermatoses of all types superior to Dalibour water. Its field of indication is broad: impetigo, ostiofolliculitis, pustular dermatoses, hypostatic dermatitis, dermatitis venenata, hyperhidrosis, bromidrosis, infected scabies, pemphigus, exfoliative dermatitis, pemphigus neonatorum, and as a deodorant in granuloma inguinale, condyloma acuminatum and severe malignancies of the skin and adnexa. In addition to being a potent antiseptic and safe for tissue cells, it is antiphlogistic, astringent and deodorizing.

In superficial folliculitis (Bockhart's impetigo, ostiofolliculitis) there is no better remedy. The solution should be dabbed on the skin about every 5 minutes. The folliculitis which causes so much distress to the individual receiving oil massages (oil folliculitis) may be prevented or promptly cured by the frequent repeated application of Dalibour water. When using it for this purpose I usually use witch hazel water or equal parts of witch hazel and peppermint water as a vehicle. While devoid of any therapeutic value this vehicle is more cooling and has a psychic influence.

5. *Silver Nitrate.* Although a potent antiseptic in dilutions up to 1:30,000, silver nitrate is a protein coagulant and a very poor antiseptic in the presence of pus and serum. For that reason it should never be used in a wet dressing in dilutions stronger than 1:10,000. When used 1:1000 as recommended by many, it forms tough adherent crusts, that seriously interfere with drainage. As in the case of potassium permanganate it is prone to promote lymphangitis. There is also valid reason to fear toxic absorption following its use on extensive surfaces. I have seen two instances of permanent argyric tattooing following its use in ivy poisoning.

6. *Brilliant Green (1:2,000).* This is a potent, nonirritating antiseptic of the aniline dye type. It is active especially against gram positive

organisms. In addition it seems to encourage epithelization. The color may be objectionable.

7. *Antibiotics.* In selecting an antibiotic for use in a wet dressing, choose one which is not given internally. You might sensitize the patient, thus possibly making it inadvisable at some future date for him to use this antibiotic to save his life. Never use any of the sulfonamides. They are potent sensitizers and many cases of severe and even fatal dermatitis exfoliativa have followed their use as wet dressings for such simple dermatoses as hypostatic dermatitis.

There are many antibiotics which lend themselves to use in wet dressings. Expense is one drawback. The second is more important; they differ from the usual chemical antiseptics in that it is essential that they come in intimate and immediate contact with the pathogen. Unfortunately, in most cutaneous infections causing inflammatory edema the pathogen is deep in the epidermis and cutis, so that the antibiotic topically is of little or no benefit. They are not used up quickly as in the case of many other antiseptics so need not be replenished as frequently. The following are the more prominent antibiotics:

Neomycin Sulfate USP. The bacterial spectrum covers many gram-positive and gram-negative organisms. Useful in impetigo, infectious eczematoid dermatitis and infected ulcers. For wet dressings 5 mg. per cubic centimeter of water are used.

Bacitracin, USP. This has essentially the same bacterial spectrum as penicillin. Use 100 units per cubic centimeter as a wet dressing.

Tyrothricin. The bacterial spectrum covers a large number of gram-positive organisms. Used locally in dilutions of 0.5 mg. per cubic centimeter. (1:2000 dilution in water).

8. *Cationic Surface-active Agents.* These make excellent, nonirritating, antiseptic wet dressings even in high dilutions. They are incompatible with soaps and alkalis but are themselves good detergents. Two of the more common ones are as follows:

Benzalkonium Chloride Solution, USP (Zephiran Chloride Solution). Its bacterial flora spectrum covers many nonsporulating bacteria and fungi. It is used 1:10,000 to 1:40,000 dilution. Although colorless, the USP permits a suitable coloring agent to be added. It is available as amorphous powder or as 1:1000 aqueous solution.

Cetyl Pyridinium Chloride (Ceepryn). This is used for wet dressings in dilutions of 1:20,000. It is commercially available as a 1:1,000 aqueous solution. Its bacterial flora spectrum is about the same as Zephiran chloride.

B. Keratoplastics

Sulfur is the only safe and effective keratoplastic useful as a wet dressing. The most effective preparation is a solution of sulfurated lime. This is official in the National Formulary and is commonly called Vleminckx' solution, but it is obtainable not without difficulty at the pharmacy. It contains active sulfide compounds in the form of calcium disulfide, calcium pentasulfide and calcium thiosulfate. The most powerful and popular method of obtaining alkaline earth sulfides, it is invaluable in the treatment of acne, especially of the deep cystic type, pyodermas and sycosis vulgaris.

A preparation equivalent to four teaspoonfuls of the official Vleminckx' solution in a pint of water is obtainable in powder form as Vlem-Dome* Powder. Gauze compresses are dipped into the material and applied to the lesions as hot as can be comfortably borne. In the treatment of pustular acne the strength of the solution may be increased by the use of less water, according to individual indications and response of the patient. Sometimes it is advisable to start with a weak mixture, applying it for a half hour once daily. Then gradually increase the strength, and apply it for two half hour sessions daily unless the skin becomes too dry.

Vleminckx' solution is incompatible with mercurials and iodides. The fumes from it will also discolor metal utensils and jewelry. It should be freshly prepared.

C. Buffer Action

Healing action is facilitated by maintaining a constant pH buffering condition on the skin. Burow's solution is a buffer which will resist pH changes.

Summary

The prime axiom of dermatology is "do no harm." The judicious use of an appropriate wet dressing, correctly applied, is the best way to preclude the development of overtreatment dermatitis. Many patients make complete recovery from a wide variety of dermatoses with appropriate wet dressings, and no subsequent therapy other than a bland oil or ointment.

STUDIES ON

Calcium Cyanamide Poisoning

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Calcium cyanamide ($CaCN_2$) is a chief component of a nitrogen fertilizer which has been widely used and has caused intoxication such as headache, vertigo, vomitus, disturbances of respiratory organs, dermatitis, and sometimes even death. The author undertook to study the biochemical characteristics of calcium cyanamide by experiments in vitro and vivo, and further by histopathological technique. The nitrogen fertilizer used here contains many other kinds of materials in addition to calcium cyanamide ($CaCN_2$), and the intoxication appears as the total effect of all harmful components in the fertilizer. We did not use refined calcium cyanamide but the fertilizer itself; so in this report, "calcium cyanamide" or "$CaCN_2$" signifies the fertilizer, a mixture of calcium cyanamide, various cyanide compounds, quick lime, carbon, etc.

In the investigation on the toxicity of calcium cyanamide, therefore, it is necessary to estimate the action of the various cyanide compounds in the fertilizer besides the effect of cyanamide itself. The estimation was performed by the use of two groups of mice, one of which was injected intraperitoneally with 100 mg of calcium cyanamide, and the other, a control group, which was given physiological saline solution. Ten minutes later the activity of cytochrome oxidase in livers was measured by a Warburg apparatus. This activity was 60.2 in calcium cyanamide group, and 64.7 in the control group; cyanide compounds might play no important role in the intoxication due to the fertilizer.

Secondly, experiments were carried out to reveal the relation between calcium cyanamide and thiol componds. G. Rodenacker, as well as some other investigators, believes that thiol compounds are useful for the prevention and treatment of calcium cyanamide poisoning because of their possible combination with calcium cyanamide. This concept was reexamined here. The solution of thiol compounds, glutathione (GSH) or cysteine, was mixed with calcium cyanamide, and with or without incubation at 37°C; the SH content in this solution was measured before and after the addition of cyanamide. No change was seen in the SH content, indicating the fact that a combination did not occur between cyanamide and thiol compounds. Two male rabbits were orally dosed with 3 gm. of calcium cyanamide and the value of GSH content was kept approximately at the initial level for eight hours. Further investigation was done by an observation on absorption spectra of GSH, calcium cyanamide and the mixture of these chemicals. λmax was 207 mμ for cyanamide, 209 mμ for GSH, and 213 mμ for the mixture, while 236 mμ for thiocarbamide accounted for the compound of thiol-radical and calcium cyanamid by some investigators. λmax of this mixture (213 mμ) approximated that of GSH and cyanamide, and its spectrum also was similar to the algebraical sum of spectra of the components. From this observation, however, it is not warranted to conclude that thiocarbamide would not be synthesized from thiol and cyanamide, because λmax of the mixture exhibited a slight tendency to move to a longer wave, and the optical density of the mixture was somewhat higher than the algebraical sum of thiol and cyanamide at 236 mμ corresponding with λmax of thiocarbamide. Upon these results the author reasons that the thiol compounds would combine with calcium cyanamide only in a slight degree.

Subsequently, experiments were performed to elucidate the mechanism on the lowered ethyl alcohol tolerance, empirically known in calcium cyanamide users, due to the cyanamide poisoning. Eighteen mice were divided into two groups; calcium-cyanamide-treated ones, and control. The former was intraperitoneally administered with 10 mg of calcium cyanamide, and two hours later both groups were intraperitoneally injected with 1 ml of 10% ethyl alcohol. Ethyl alcohol content in the brain, determined ten minutes after the ethyl alcohol injection, was 340 mg per 100 gm. of tissue for the treated group and 334 mg for control. Male rabbits were also divided into two such groups, and they were investigated on the contents of ethyl alcohol and its metabolite, acetaldehyde, in the blood after ethyl alcohol injection. The results are illustrated in Figures 1 and 2. It is obvious that calcium cyanamide does not accelerate the accumulation of ethyl alcohol, but that of acetaldehyde in vivo after ethyl alcohol administration. This suggests that calcium cyanamide may inhibit the process in which acetaldehyde is metabolized to acetic acid in vivo. Succeeding experiments were, therefore, devoted to studies on the relation between acetaldehyde oxidase and calcium cyanamide. Tissue homogenate was used as a source of acetaldehyde oxidase, and the ac-

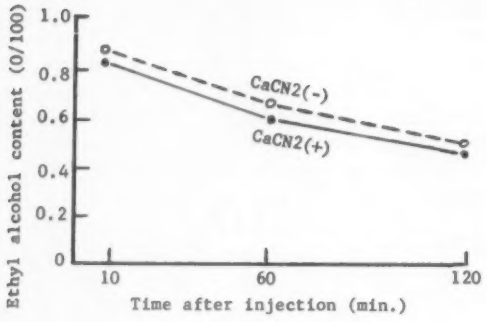

Figure 1
Ethyl alcohol content of rabbit blood following intravenous injection of ethyl alcohol.

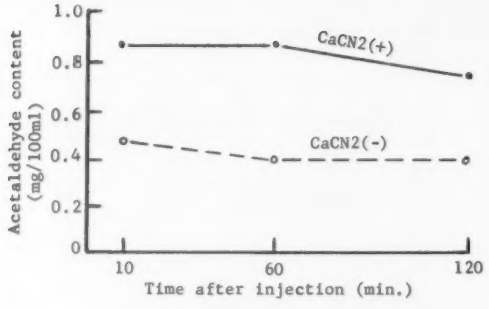

Figure 2
Acetaldehyde content of rabbit blood following intravenous injection of ethyl alcohol.

tivity of the enzyme for the normal mouse was shown in the following order: liver, 2.45; heart, 0.56; kidney, 0.54; spleen, 0.30; brain, 0.26; and lung, 0.11 γ/min/gm. of tissue. Throughout the below-described experiments, liver homogenate was used as a source of the enzyme, because its activity is highest of all tissues. Inhibition of acetaldehyde oxidase by calcium cyanamide was examined in vitro, adding the poison to the system for determination of the activity of the enzyme, and it was found that there was an 83% inhibition by 1 mg of the inhibitor per ml in the final concentration, 45% by 0.1 mg per ml, and 10% by 0.01 mg per ml.

Subsequently, the effect of vitamins and detoxicants was investigated on the reactivation of acetaldehyde oxidase inhibited by 0.1 mg of calcium cyanamide per ml in the final concentration. No effect was found for vitamin B_1, vitamin B_6, glucuronic acid and cysteine, while considerable efficacy was perceived with the addition of vitamin C and GSH: when 2 mg of these chemicals per ml of the system for determination was added, the reactivation rate was 43% for vitamin C and 36% for GSH. The effect of GSH may be not due to the combination with cyanamide by its thiol radical, but to its high biological activity, because there was no effect with cysteine having a like thiol radical, and this is in concord with the results of the antecedent experiments on the combination between cyanamide and thiol compounds.

In addition, an experiment was carried out on the basis of speculation that there would likely exist a correlation between the activity of acetaldehyde oxidase and alcohol tolerance. The alcohol tolerance was determined as follows: 13 mice were intraperitoneally injected with 10% ethyl alcohol in the rate of 1/15 of the body weight. A few minutes afterward, mice lay down due to the acute intoxication by ethyl alcohol. They recovered from the intoxication according to the lapse of time, and stood up again. The alcohol tolerance was determined for each mouse measuring the time from falling to standing. It may be considered here that the shorter is the recovery time, the higher is the alcohol tolerance. At one week after this investigation, the activity of acetaldehyde oxidase in liver was examined for each mouse and the author obtained results showing a high correlation between the activity of acetaldehyde oxidase and the alcohol tolerance (Figure 3).

Further study was undertaken on the lability of the activity of acetaldehyde oxidase. Fifteen male mice, divided into three groups, nonfeeding for one and three days and control, were studied for the activity of acetaldehyde oxidase in liver. This activity was 2.96 in control group, 2.26 in nonfeeding for one day, and 1.15 in nonfeeding for the three day group. We conclude the activity of acetaldehyde oxidase is labile and is remarkably lowered by nonfeeding. Similar experiments were performed on the activity of cytochrome oxidase, and the influence of nonfeeding was not perceived in this enzyme.

According to the experimental results on acetaldehyde oxidase, calcium cyanamide does not accelerate the accumulation of ethyl alcohol, but that of acetaldehyde in vivo after administra-

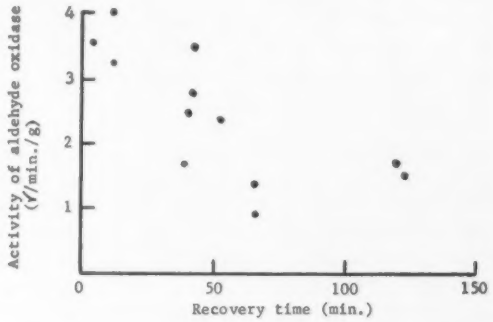

Figure 3
Correlation between the activity of aldehyde oxidase and the alcohol tolerance.

CONCENTRATION OF NITROGEN COMPOUNDS IN BLOOD AND URINE OF RABBITS BEFORE AND AFTER THE ADMINISTRATION OF CALCIUM CYANAMIDE

	$CaCN_2$ dosed (g)	Uric acid (mg%)		Urea nitrogen (mg%)		Thiocyanate (mg%)		Ammonia (mg%)		Total nitrogen (g%)	
		Before	After	Before	After	Before	After	Before	After	Before	After
Blood	0.5	3.5	2.7	29.0	30.8	0.35	0.49	—	—	2.91	2.99
	1	3.0	3.0	25.0	28.4	0.59	0.66	—	—	3.14	3.25
	1.5	3.7	4.0	17.5	17.7	0.40	0.41	0.81	1.23	2.73	2.77
	3	3.2	3.5	27.8	37.7	0.52	1.01	0.99	1.73	2.80	3.36
Urine	0.5	34	35	1,270	1,240	0.68	0.62	44	55	1.44	1.50
	1	28	37	1,190	1,130	1.21	1.29	37	67	1.25	1.31
	1.5	14	24	1,250	1,600	0.93	1.50	51	103	1.49	1.81
	3	19	44	1,340	1,600	0.83	1.37	42	120	1.55	1.89

tion of ethyl alcohol. Jacobsen also pointed out the increase of acetaldehyde in cyanamide poisoning, and he concluded that the enzymes which control the combustion of ethyl alcohol in the liver are affected by calcium cyanamide. Under the experiments both on ethyl alcohol and acetaldehyde content in blood and on inhibitory effect of cyanamide in vitro, it is acetaldehyde oxidase that may be affected in this poisoning. Furthermore, this enzyme is labile in its activity and has a high correlation with ethyl alcohol tolerance. The reason why the activity of cytochrome oxidase is not so labile as that of acetaldehyde oxidase is perhaps the fact that the former is an essentially important enzyme for life itself, while the latter is not an important enzyme, substitutive by xanthine oxidase. It was already mentioned that the activity of acetaldehyde oxidase is most remarkable in liver, and according to the author's histopathological experiment in which a rabbit was injected in the stomach with calcium cyanamide, the liver is an organ easily damaged by cyanamide; the cell infiltration was found in the Glisson's capsule, and the Kupffer's cells were proliferous and hypertrophic in the liver, but no change was seen in brain, heart, lungs, spleen and kidneys. In summary, one of the mechanisms of the lowered ethyl alcohol tolerance by cyanamide may be due to the accumulation of acetaldehyde caused by inhibition of acetaldehyde oxidase, which is labile, concentrated in liver, and has a profound relation to ethyl alcohol tolerance. This explanation is, however, not sufficient to clarify the mechanism of the lowered ethyl alcohol tolerance in cyanamide poisoning: the acetaldehyde in blood is only 1/100 of the ethyl alcohol in the previous experiments, although the former is 10 times more toxic than the latter according to the author's investigation. It is not then allowable to exaggerate the effect of acetaldehyde, but it is necessary to seek any other factors, too, as the author has remarked in the course of studying the metabolism of calcium cyanamide.

Calcium cyanamide was administered into the stomachs of male rabbits and the concentration of urea, uric acid, thiocyanate, ammonia and total nitrogen in the blood and urine were determined. The results are shown in the table. Biochemical changes in the body are so dynamic and complex that it is not always right to conclude, for example, that the increased amount of urea in blood and urine must be attributed to calcium cyanamide dosed to the animal, but we may recognize in this way the outline of metabolism of calcium cyanamide. Two important conclusions are derived from this study on the metabolism: one is that the greater amount of calcium cyanamide is excreted in urine as urea, and the other is that as for the ratio of the concentration of nitrogen compounds after the administration compared to that before administration, its highest value is seen for ammonia. The process producing ammonia from ingested calcium cyanamide is considered as follows:

$$CaCN_2+2HCl \rightarrow H_2CN_2+CaCl_2;$$
$$H_2CN_2+H_2O \rightarrow HCNO+NH_3;$$
$$HCNO+H_2O \rightarrow CO_2+NH_3$$

In general, the ammonia produced in vivo is rapidly synthesized to urea in the normal metabolism. This is shown by the result that there was no significant difference for the concentrations of ammonia in the blood between, before, and after ingestion of amino acids. On the contrary, the synthesis of urea from ammonia in liver may be inhibited by calcium cyanamide that damages the organ, and ammonia is accumulated in the organisms. Thus, calcium cyanamide is not only a producing source of ammonia but also an inhibitor in the metabolism of ammonia. Besides ammonia, there is seen also the production of hydrocyanic acid. Therefore, the increase of such poisonous compounds as ammonia and hydrocyanic acid may lower the resistance of the body, and further may lower the alcohol tolerance in calcium cyanamide poisoning.

The author wishes to express his profound acknowledgement to Prof. Dr. M. Nishio who has given kind advice at all times. For detailed data of this work, see the complete study in *Japanese Journal of the Nation's Health* Vol. 28, p. 263-283, 1959.

Are You Getting Through?

• W. C. BOYD
Division of Weights and Measures
Indiana State Board of Health
Indianapolis

The need for the art of good communication by no means is limited to any one profession, to any one industry, or to any one nation. The requirements of precision in medicine to an extraordinary extent demand good communication. The majority of the misadventures in medicine stem not from poor judgment on the part of the physician or nurse but from some break in good communication somewhere along the line, whether it be oral or written. Good communication is just as easy as faulty communication and many times easier. The hope is that this nontechnical presentation itself may prove to possess and to exhibit some of the qualities of simple but effective communication.

What is communication? One author has said, "Communication is 'saving' dressed up in its Sunday best." The dictionary gives several meanings: "to share in"; "to give to another"; or "the exchange of thoughts, opinions and ideas."

In all, there are 27 methods of exchanging information. Most of these are limited substitutes for face-to-face discussion. In the early days of American business, individual enterprises were small enough to permit man-to-man discussion of personal and administrative problems. Today, due to business and industrial expansion, these personal discussions are not usually possible or practical. Consequently, communications now involve more time and people than does any other single administrative activity. In fact, the average administrator spends 90% of his time talking, writing, or listening; all of which are a part of the communication process.

In industrial health work, as well as in other fields, one of the basic problems is to convey our ideas, thoughts, and information in such a way that our message will be understood as we intend it to be understood. In personal discussions of a mutual problem, we not only hear the message, but we can depend on the speaker's tone of voice, inflections, and actions to convey his intent. If misunderstanding or misconception occurs, we can continue the discussion until a mutual understanding exists.

Conversely, written communications are similar to the telegraph. Once transmitted, the message is beyond the sender's control. What the message means to the person receiving it, the attention he gives it, and his response to it will depend on the effectiveness of the content and the method of transmission. The effectiveness of our day-to-day communications can be improved simply by applying certain practical principles that are the result of extensive research in the field of human motivation.

Each individual is the center of a communication network. People and things constantly seek his attention. You want him to pay attention to your message, but you can't force him to do so if he doesn't want to; not even if you pay him. Many people go to sleep or "day dream" on company time. There has to be a reason for the person to pay attention — an incentive — and there can be.

Social scientists have proved conclusively that every individual, regardless of his station in life, is motivated by four basic urges or "rewards." These are:

(1) *Social Response:* The desire to belong; to be a member of the team.
(2) *Security:* A job, a pay check, freedom from fear, doubt, or want.
(3) *Recognition:* Self-esteem, pride, the desire to be "somebody"; also called "ego."
(4) *New Experience:* A desire for learning or knowledge, a chance to stimulate his interests or abilities.

If your message appeals to one or more of these motivations, it will get attention.

People need to understand a message before they can give it their best efforts. They need to know the "why" as well as the "what." The old idea of "do-this-or-else" went out of existence with the realization that people are different from machines. Correspondence is a substitute for a personal meeting. When you write a letter, a memorandum, or a bulletin, you want it to do

From *The Monthly Bulletin*, Indiana State Board of Health, September, 1959.

what you would do if you called in person.

If your correspondence is to represent you in a favorable, advantageous manner, it must be:

(a) *Correct:* In form and content. An inaccurate message does real harm and it gives official status to error.

(b) *Definite:* To the degree that the reader will get the intended meaning, and no other.

(c) *Complete:* In that it includes every essential point, and does the job thoroughly; and,

(d) *Courteous:* In that it reveals, by word and tone, true friendliness and a desire to understand the other person's viewpoint.

Communication has been discussed as a means of exchanging information, but there is another important function; that of reporting to interested parties what has been done. The reporting function is, in general, not as well understood as the exchange of information. The majority of reports are written out of sheer compulsion, usually because of a legal or moral obligation to count up the score to satisfy a supervisor, a budget committee, or perhaps, the general public. The best that can be said for such reports is that they fulfill the limited purpose of setting out in print a bare record of facts.

Business and governmental organizations have grown so large that there are few, if any, of us who do not have supervisors and associates to whom we must report our activities. Keeping these people informed does *not* mean that we are to be a substitute for the daily newspaper; neither are we expected to be the secret agent of the organization "grapevine." It *does* mean that our supervisor should hear about our success or about any unusual service we have been able to give to associates, the organization, or to the public. It also means that he should hear about our errors directly from us and not from some other source.

Oral reports have only one real advantage. This is the opportunity for all persons concerned to discuss the problem personally. The principal objection to oral reports is that they do not provide a permanent record, also, that it is difficult to transmit correctly the report to others.

On the other hand, a well-written report that includes statistics, accompanied by an interesting narrative, can teach new and important facts about conditions that give your office a reason for existing. It can help you gain the support of your supervisors and the public. Your report can become a fighting document — a chance to point out your needs as facts reveal them. Finally, it is your best opportunity to tell about your work; to appraise its accomplishments and to study the reason for its failures. Properly prepared, our reports become not an end to themselves, but a powerful means toward the objectives we wish to attain.

How well a message accomplishes its purpose is a true test of the effectiveness of the content. Words can be either an aid or a barrier to understanding. Words that are exact, appropriate, simple, clear, and direct promote a better understanding of the message.

Technical words have become such a definite part of the common language of society that people are finding it impossible to express their thoughts in a simple, effective, and understandable manner. For the most part, technical words and phrases should be avoided unless the subject or occasion warrants their use. When such words or phrases must be used, they should be defined.

Good communication is not "a one-way street." It has three dimensions — up, down, and across. The "downward" path is, in general, well used, but unfortunately, the "upward" and "across" paths are relatively untrodden. Effective coordination of any organization's efforts depends on information traveling up and down the administrative lines as well as across lines of authority. An established pattern or plan for the exchange of information will permit the message to move step by step through the organization until it reaches the person or persons who can take the necessary action or make the necessary decision. Such a plan will help pinpoint individual responsibility, reduce error, and prevent misunderstanding. Furthermore, it will give each individual an improved picture of the work, accomplishments, and problems of those with whom he works.

Let us not forget that, actually, there is no such thing as a "one-way" communication. An unanswered letter, failure to supply requested information, or failure to follow instructions, each send "silent messages." These "silent messages" may be due to lack of interest, skill, or knowledge; failure to take time to understand the message; or other interests that distort the message. All of these "blocks" to good communications can be eliminated by willingness to learn, attention to the subject, and by applying one's self to the task.

Group communications are important to bolster our understanding of mutual problems. These group activities, such as semi-annual and annual conferences, committee projects, and training schools, may be utilized to pass along new information concerning developments in our chosen field; to discuss changes in existing policies and plans; or for the pooling of ideas to meet a common problem.

Group Insurance — A Mounting Production Cost was discussed at the Management Conference by: (seated, left to right) E. T. Klassen and Dr. J. F. McCahan; (standing, left to right) R. R. Shinn, and Dr. W. P. Yant, Board Chairman of I-H-F, who was Chairman for this discussion.

A panel discussion on "Methods of Measuring Work Capabilities of the Individual" and "Usefulness of Procedures of Health Examinations" were the topics for the Medical Conference. Participants were: (left to right) Dr. W. H. M. Morris, Dr. L. W. Gregg, Dr. J. P. Hughes (Chairman), Dr. H. S. Belding, Dr. W. D. David, and Dr. R. B. Chevalier.

INDUSTRIAL HYGIENE FOUNDATION

25th Annual Meeting Mellon Institute, Pittsburgh

The Industrial Hygiene Foundation held its 25th Annual Meeting at the Mellon Institute, Pittsburgh, October 26 and 27, 1960.

"Group Insurance — A Mounting Production Cost" was discussed by three speakers: E. T. Klassen, American Can Co., New York City; R. R. Shinn, Metropolitan Life Insurance Co., New York City; and Dr. J. F. McCahan, Western Electric Co., Chicago. Elimination of abuses and establishment of better controls were advocated as ways to reduce the increasingly higher costs of insurance and employee health care.

One of the most troublesome problems facing the public and the consumer-chemicals industry was also presented at a Management Conference. Dr. Anna M. Baetjer of Johns Hopkins University was the Moderator and three speakers discussed the "Effects of Food Additives Legislation on Food Technology." They were: K. E. Mulford, Atlas Powder Co., Wilmington, Delaware; L. A. Coleman, Allied Chemical Corp., New York City; and D. B. Hand, Cornell University. Highlights of the session were talks on two aspects of the problem. One of these was the heavy expense involved in long-term research necessary to investigate the safety of new materials. Expenditures of $500,000 by a chemical manufacturer are not uncommon in the effort to prove that a substance is safe, with no guarantee that the government will approve use of the material even though data appear to be favorable. Another aspect of the problem is the effect upon the public of arbitrary enforcement of food-additives legislation. Under such enforcement, government banning of substances used in modern insecticides could deprive the public of agricultural products made plentiful because of the efficiency of these same pest controls.

The final topic at the Management Conference was "Standards for Air Quality," a discussion of trends in municipal air pollution legislation. The speaker was V. G. MacKenzie, Chief of the Division of Air Pollution, U. S. Public Health Service. Mr. MacKenzie is a coordinator for the government on air-quality standards. The question of whether such standards are needed, whether they can be set up, and whether they are practical was considered. Current efforts of the city of Los Angeles to establish standards, if successful, could have a profound effect upon other cities and states.

The Joint Medical-Legal Conference took the form of a panel discussion. "Emphysema, Pneumoconioses, and Compensation" was the subject of the important forum on the problem of dust pulmonary diseases which still persist despite medical advances. The Moderator was V. P. Ahearn, National Industrial Sand Association, and the panel members were: Dr. O. A. Sander, Marquette University; Dr. A. J. Vorwald, Wayne State University; Dr. G. W. Wright, Saint Luke's Hospital, Cleveland; and W. C. Hartman, Squire, Sanders & Dempsey, Cleveland.

A panel discussion on "Methods of Measuring Work Capabilities of the Individual" was the major part of the Medical Conference. Dr. J. P. Hughes of Kaiser Aluminum and Chemical Corp. acted as Moderator and Dr. L. W. Gregg, Car-

negie Institute of Technology, discussed the prediction of human tolerance to stresses of specific jobs. Factors such as high blood pressure and emotional stability were considered in stresses such as those experienced by the astronaut, the worker in a hot steel mill, and the white collar employee. A related talk dealing with a unique procedure was presented by Dr. H. S. Belding, Graduate School of Public Health, University of Pittsburgh. Dr. Belding described instrumentation that makes possible remote monitoring of a worker's physical and psychological strains, without interfering with the worker's activity. Dr. W. H. M. Morris of Purdue University spoke on the "Physiological Approaches to Evaluational Capabilities." The "Usefulness of Procedures of Health Examinations" was presented by Dr. W. D. David of the U. S. Public Health Service.

A panel discussion on "Effects of Food Additives Legislation on Food Technology" at the Management Conference: (left to right) L. A. Coleman, K. E. Mulford, Dr. Anna M. Baetjer (Moderator), D. B. Hand, and Dr. K. W. Smith (Chairman), Johns-Manville Corp.

Aspects of workmen's compensation constituted the entire program of the Legal Conference. An important discussion of "Legislative Developments in Workmen's Compensation for the Year 1960" was presented by Andrew Kalmykow of the Association of Casualty and Surety Companies, followed by a discussion by D. W. Stitt, Continental Can Company. A second talk by H. F. McNiece, St. John's University School of Law, dealt with the legal basis for awards that have been made in specific cardiac cases. Emphasis was given to cases involving attacks based on mental strain. Mr. O. T. Clayton, Esso Standard Oil Company, discussed "Problems Presented to Directors of Departments of Industrial Relations in the Handling of Workmen's Compensation Claims."

A highlight of the Engineering Conference was a discussion of the expansion of city zoning regulations to control industrial plant locations from the standpoints of dust, smoke, noise, vibration, illumination nuisance, and toxic-matter emission, by W. T. Ingram, American Society of Civil Engineers. Other topics included: "Acceptable Equipment and Procedures for Evaluating Air Pollution Problems" by R. O. McCaldin, U. S. Public Health Service; "Benefits of Central Gas Cleaning Facilities" by C. A. Bishop and G. A. Howell, both of United States Steel Corp.; "How Can We Achieve Better Operation of Dust Control Systems" by K. J. Caplan, Mallinckrodt Chemical Works; "Recent Welding Practices — Naval Facilities" by C. P. Bergtholdt, U. S. Naval Weapons Plant; "Legal Considerations Involved in Developing Precautionary Labels" by J. D. Kittelton, Manufacturing Chemists' Association; and "A Progress Report on In-Plant Hazard Identification Systems" by J. J. Duggan, of the Union Carbide Chemicals Company.

Recent developments in industrial hygiene were

A panel discussion on "Emphysema, Pneumoconioses and Compensation" at the Joint Medical-Legal Conference by: (seated, left to right) T. C. Waters, Miles & Stockbridge; V. P. Ahearn (Moderator), W. C. Hartman, (standing, left to right) Dr. A. J. Vorwald, Dr. G. W. Wright, and Dr. O. A. Sander.

discussed by H. W. Speicher, Westinghouse Electric Corp.; R. H. Blackmer, General Electric Co.; N. G. White, Shell Chemical Corp.; R. J. Wright, American Air Filter Co.; and R. T. Pring, Dracco Division of Fuller Company.

"Chemical Antidotes for Radiation Injury" was discussed by Dr. John Doull, USAF Radiation Laboratory, University of Chicago, at the Chemical-Toxicological Conference. Dr. Doull emphasized the necessity for awareness of hazards in the development of new rocket fuels "from test-tube to launching pad." Dr. J. A. Clements, Army Chemical Center, presented a paper on "Pulmonary Edema and the Permeability of the Alveolar Membranes"; and Dr. C. J. Kensler, Arthur D. Little, Inc., followed with his talk on "Toxicologic Mechanisms at the Cellular Level." Fuels and their combustion products were discussed by: J. F. Morgan, Pennsylvania Railroad (diesel fuels); E. B. Rifkin, Ethyl Corporation (missile fuels); and H. M. Bowman, Reaction Motors Division of Thiokol Chemical Corporation (rocket fuels).

Books

Principles of Surgical Practice

Emanuel Marcus and Leo M. Zimmerman. $12.50. Pp. 430, 58 illustrations. McGraw-Hill Book Company, Inc., New York, 1960.

The undergraduate medical student's curriculum in the surgical fields must make him aware of the application of what he learned in the basic sciences to the diagnosis and treatment of the sick or injured patient. Didactic teaching programs decrease with the emphasis on bedside teaching, as the student advances. A general introductory course in surgery bridges the gap between the basic sciences or classroom instruction, and the study of the patient; this book has been prepared to serve that purpose.

It does not attempt to cover the techniques of surgical operations. It is admirably adapted to the junior and senior medical student's needs, for it outlines the essential anatomical, physiological, pathological, and other backgrounds in the etiology, symptoms, diagnosis, and treatment of the common disorders now amenable to surgical attack. By so doing, it can serve a purpose for the general practitioner and the specialist, as a reference book of fundamentals which may have been forgotten.

This reviewer was particularly impressed by the chapters on parenteral alimentation, peripheral vascular diseases, the thyroid, thoracic injuries, the biliary system, and hernia. The illustrations are few (58) but are well selected. More will probably appear in future editions. The text is well written and the material thoughtfully chosen. At the end of each chapter is a summary of principles and a small list of references for further study. The authors have accomplished their objective of preparing a book which can be read with profit by all advanced medical students and interns, and by most clinicians.

—CHESTER C. GUY, M.D.

You Can Prevent Illness

Edward R. Pinckney, M.D. $3.50. Pp. 157. J. B. Lippincott Company, Philadelphia, 1960.

Every day some hundreds of physicians go about in harassment seeking the perfect small gift for a patient or for a social friend. Every day even more hundreds of persons close to physicians would like to query "Where can I find a small book that will tell me concisely about health, diseases and how to prevent diseases and what I can do about all such—a book that I can believe?" The answer to both these perplexities is this publication by Dr. Pinckney who writes engagingly about vaccines and vacations, about travels and tasks, about dentists and doctors—and every word may be believed and every word may be understood by all. This is no "home medical consultant" with platitudes about plenty of sleep or about espousing fads in foods.

The next best thing to having the physician sitting alongside in a comfortable chair engaged in earnest conversation is this book that speaks with clarity and understanding of what many thousands of readers want to know about their health and how to preserve it.

The Merck Index

Paul G. Stecher, Editor. Seventh edition. $12.00. Pp. 1600. Merck & Co., Inc., Rahway, N. J., 1960.

For many industrial physicians and industrial hygienists, consultation of the *Merck Index* is the initial step in the investigation of any unknown or uncertain substance. It is as indispensable as salt on the dining room table at mealtime.

The Seventh Edition just out is more serviceable than ever. It contains about 1,600 pages of text covering nearly 10,000 descriptions of individual substances, more than 3,300 structural formulas, and about 30,000 names of chemicals and drugs alphabetically arranged and cross-indexed.

The previous edition of the *Merck Index* was published in 1952 and 100,000 copies were purchased before the supply was exhausted during 1958. The new edition includes extensive scientific advances during the past eight years.

An outstanding feature of the new book is a separate and greatly expanded cross-index section of more than 30,000 names. This enables the user of the book to locate a particular chemical description by page number regardless of whether he knows only the generic name, brand name or systematic chemical name for a substance.

A special section lists more than 400 organic "Name" reactions with original and review references, together with a description and structural representation of each reaction. There is an up-to-date periodic table arranged in accordance with the latest concepts of nuclear science, a table of international atomic weights and close to 300 pages of appendices on such subjects as chromatographic absorbents, calories in foods, Russian alphabet, biological units, coal-tar colors, thermometric equivalents, antifreeze mixtures, refractive index of liquids, saturated solutions, radioactive isotopes, percentage solution tables for apothecaries, isotonic solutions

and atomic weights and their multiples and logs.

Each main entry indicates the preferred chemical name or other chemical names, common or popular names, generic and trade names. Trademark listing is as complete as possible and thoroughly cross-indexed.

Properties and sources of substances, preceded by methods of preparation, with literature references, are given, followed by an indication of uses, whether industrial, medical or veterinary.

Hazards, toxicities, and other safety factors are mentioned.

Indication is given of commercial availability and grades, such as U.S.P., N.F., and reagent.

Empirical formulas, molecular weights, and percentage composition are given for all chemicals having a definite structure.

As to the sales price of this book, it is the best bargain at any book store. It may be guessed that every copy of the first 10,000 printed cost four times the going price available to all.

Principles of Disability Evaluation

Wilmer Cauthorn Smith. $7.00. Pp. 210. J. B. Lippincott Company, Philadelphia, 1959.

This book is based on the practices of the Oregon Industrial Accident Commission. Some books on disability evaluation approach mathematical precision providing almost exactness for every disability situation requiring rating. Although this book contains much exactness foremostly it is devoted to the theory and philosophy of disability appraisal. With admirable restraint the author over and over urges that many disabilities do not lend themselves to any set formula. This is made clear in one excerpt:

"The only practical approach which provides uniformity and lies within the realm of medical science is to evaluate the occupational handicap occasioned by the damage to the unscheduled organs or parts. If the examiner is to be realistic and remain within the field of medicine, this means that his evaluation of unscheduled disability is in reality a measure of occupational handicap. If occupational handicap has not resulted, medically ratable disability is not present. Should it be the desire of society that disability compensation be allowed for the loss of one testicle, one ovary, one kidney or the spleen, these organs must be made the subject of statutory scheduling. This would provide general acceptance and the necessary uniformity. The physician is justified in evaluating only the occupational handicap; he is not qualified to place arbitrarily a monetary value on organs or parts."

That disability rater seeking an easy way out for his tasks will find small comfort in this text. The masters and the would-be masters in this trying aspect of medicine who place high values on sound judgment will discover invigorating provocation in every section of this text.

Minor Hand Injuries (The Care of)

Adrian E. Flatt. $9.50. Pp. 266, illustrated. The C. V. Mosby Co., St. Louis, 1959.

To be intimate and confidential in a scientific book without being lordly and condescending, is an art. This author is that kind of an artist. He is ever the youthful grandfather and never the Dutch uncle. To him, apart from the trivial surface injuries, there are no minor hand injuries. He detests that term as it appears in his title; but almost perforce he is compelled to use it. He knows that few "minor" hand injuries reach the true surgeon and that the care rendered all too often is not the best. It is his thesis that the best is the simplest. So he pursues that thesis through such chapters as: Functional Anatomy, Principles of Care, Surgical Techniques, Classification and Examination of Injuries, Wounds of the Skin, Injuries to the Nail, Pulp Loss, Amputations, Crush Injuries, Tendon Injuries, Joint Injuries, Fractures, Burns, Infections and Miscellaneous Injuries and Wounds.

The old complaint of the general practitioner and the industrial physician is that books tell him everything except what he needs to know at a given moment. That charge may not be leveled at this book. It provides all that the general practitioner, who does a little surgery, needs to know in the care of "minor" hand injuries, in a manner that inspires confidence. The salty flavor of this will become apparent in one paragraph in the preface:

"Probably only the author, the publisher, and the more conscientious reviewers will attempt to read straight through the second section. My publisher has no choice, but to the reviewers, I would say, please do not attempt this feat. You will be bored by repetition. The text has been planned to be dipped into as the situation warrants. In times of stress, the index and chapter headings are usually searched for appropriate help, and it is hoped that the repetition of material in these sections will obviate the infuriating necessity for repeated back reference to earlier pages."

This reviewer who is not smugly conscientious, but who has been taken into camp by the author, ignored that injunction and read the book in its entirety, and he is no surgeon — hand or otherwise.

INDUSTRIAL MEDICINE AND SURGERY

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January, 1961

VOLUME XXX

NUMBER 1

The Disappearance Of Industrial Medicine

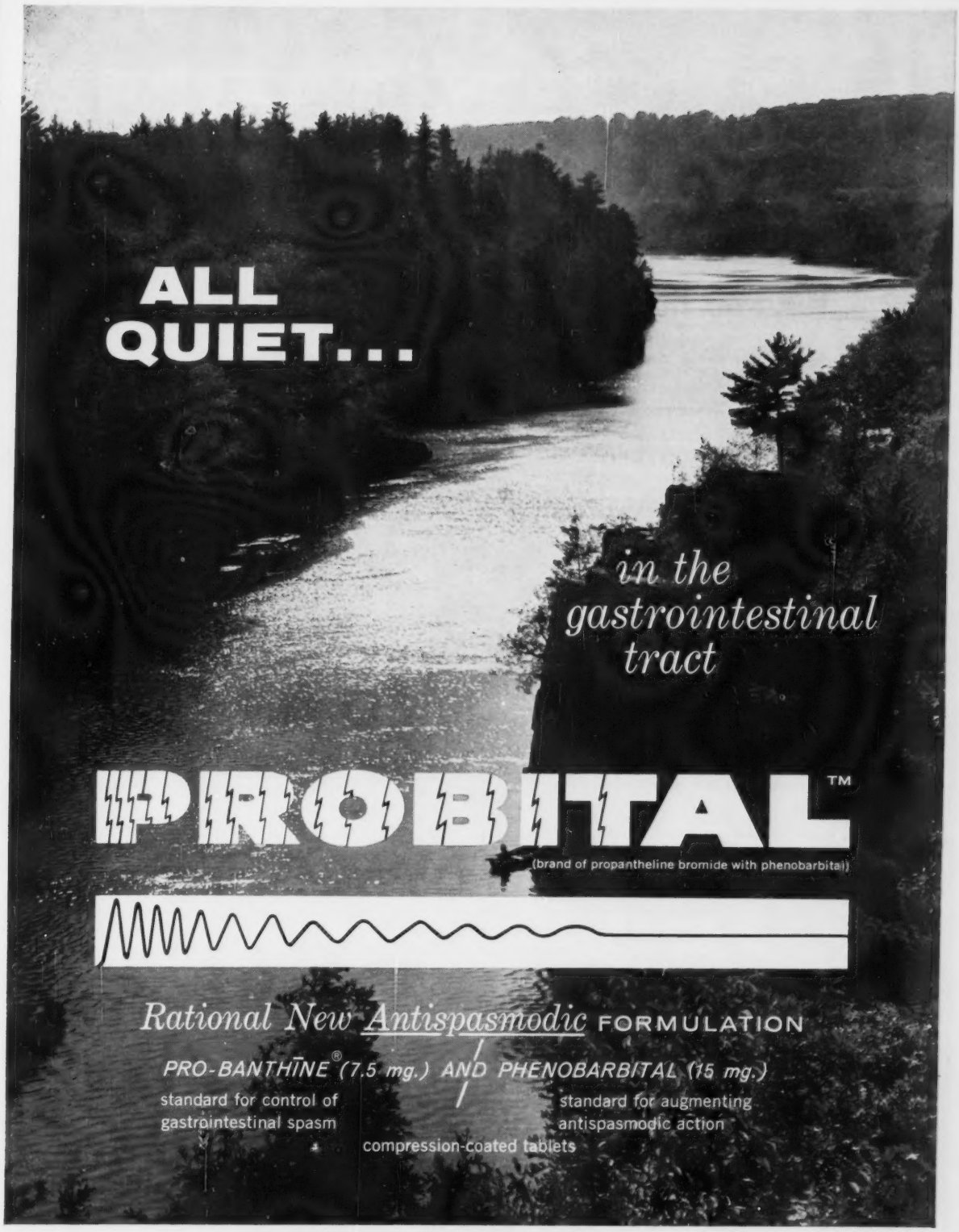
At the time of the recent International Congress on Occupational Health in New York, one speaker, a representative of a labor organization, predicted the disappearance of industrial medicine in the foreseeable future. Perhaps no other single statement attracted more attention. Circumstances did not arise affording opportunity to interrogate that speaker as to the significance of his startling prophecy. As a possibility, he may have meant to indicate that with the shifting ramifications of medical care, the term "Industrial Medicine" might be lost. If true, that is a matter of semantics and is not a matter of the industrial worker's needs for medical surveillance.

It is more likely that the intent was to assent that with the accomplishments of the plant designers, the plant engineers and the industrial hygienists, industrial traumatic injuries and characteristic occupational diseases might fade from medical requirements. That is a near possibility.

Already in the United States, the characteristic occupational diseases are on the wane and so much so that trainees in medical centers seldom see clinical cases of states that a few years ago were commonplace. Yet there is no sound reason to expect that at any early time, occupational injuries and diseases will vanish. Notably this is true for smaller establishments wherein no medical services exist.

The good industrial medical program is far broader than industrial trauma and occupational diseases. Even if all such emergency requirements did fade away, the good medical program would be little altered. Foremostly industrial medicine is concerned with prevention; prevention through the maintenance of outstanding hygienic ministrations; prevention through physical examinations; prevention through proper placement; prevention through counseling; prevention through education; prevention through research; prevention through fitting the industrial medical department into the community.

Pronouncement of the impending death of industrial medicine requires an eyesight and a mind-sight not now known to exist. No grave is being dug.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References: 1. Brownlee, George: A Comparison of the Antipyretic Activity and Toxicity of Phenacetin and Aspirin, Quarterly J. of Pharmacy and Pharmacology, 10:609-620. 2. Goodman, Louis S. and Gilman, Alfred: The Pharmacological Basis of Therapeutics, sec. ed. 1955.

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COMMENT AND OPINION

Abnormal Hemoglobin Retards Malaria

The gene that produces red blood cells of abnormal structure—the so-called sickle cells which, in sickle cell anemia, cause death—serve also to protect bearers from malaria.

This is the best known example of natural selection in human populations, according to a paper read at a New York Academy of Sciences conference on genetics by Dr. Anthony C. Allison of the National Institute for Medical Research, London, England.

As many as 4% of all babies born among some tribes studied in East Africa where malaria is endemic bear two genes for sickle cells. Most of them die before adolescence because of sickle cell anemia. However, in the same areas, up to 40% of all babies are born with a single gene for sickle cell, and are rendered thereby relatively immune to malaria.

The protection is explained by the fact that the malaria parasite cannot use efficiently the hemoglobin in the sickle cell which differs only slightly from that in the normal red blood cell. This is not truly an immunological phenomenon but an example of protein resistance.

Another example of genetic resistance to malaria has also recently been found among the same Africans. This is a single gene which causes the body to produce a deficiency of the red blood cell enzyme glucose-6-phosphate dehydrogenase which is essential to the life cycle of the malaria parasite. These two protective genes are effective chiefly in childhood. So universal is malaria that even those who lack either gene acquire immunity by the time they reach adulthood.

Insurance Abuses Hit

"Voluntary health insurance is a fine example of the ability of industry to forestall any movement toward the welfare state," W. L. Hogue, M.D., Medical Director of The Firestone Tire & Rubber Company told the members of the Polk County Medical Society in Des Moines, Iowa.

"Important involved groups in the economy are committed to making our health insurance program work well enough that a comprehensive state program is unnecessary," he continued, "and it is in the fields of costs and responsibility that we are concerned."

Dr. Hogue, the principal speaker at a dinner meeting held at the Firestone plant, pointed out that the burden of the costs of medical care had been shifted from individuals to organizations and that the total amount of money available for medical services had increased many fold. He emphasized that all private expenditures for medical care amount to $15 billion annually, according to the Department of Health, Education, and Welfare. This amounts to as much as two thirds of the value of all residential construction in a given year.

He warned that, "All groups involved have definite and positive obligations to the community and to the economy to see that increased costs, if any, are real and not inflationary."

Dr. Hogue emphasized that health insurance benefits are up 8% this year compared with last year, and he stated that at Firestone the cost per man hour worked for hospital and surgical benefits has practically doubled since 1956 and that sickness and accident benefits have increased 40%. He explained that when these increases in costs of health and hospital care are not in line with general index increases, "We must look to our procedure."

"The Medical Care Index lagged behind the Consumer Price Index from 1935 to 1946," he stated, "then increased at approximately the same rate until 1949, after which it has increased at a substantially faster rate."

He warned against the abuses of health insurance that tend to inflate the benefit costs. Among these, he enumerated the performance of medical services included in the contract but which would not be performed if the

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patient were not insured; excessive, multiple visits by doctors; unnecessary hospitalization; higher fees than for uninsured; excessive hospital stay; excessive, lengthened convalescence; and repetitive use of test and analysis techniques.

"Industrially speaking," said Doctor Hogue, "it must be remembered that the cost of health insurance is a cost of doing business.

Polio Shots

About 93 million people, representing 60% of the population under 60 years of age, have had at least one dose of polio vaccine, according to new estimates announced by Surgeon General Leroy E. Burney of the Public Health Service.

The estimates are based on a sample survey conducted by the Bureau of the Census in September, 1960. A similar survey made in September, 1959, showed that a total of 85 million people, or 56% of the population under 60, had had at least one dose of vaccine.

Most of the vaccinations given in 1960 were booster shots. About 25% of the population have now had four or more doses as compared to 14.2% in 1959.

Although infants and children under five have accounted for most paralytic polio cases in recent years, 20% of these children have had no vaccine. This is but a slight improvement over the 1959 situation when 23% had received no vaccine. The proportion of preschool children and infants with three or more doses increased, from 54% in 1959 to 62% in 1960. Of the 8 million children in this age group who remain inadequately protected, 3.8 million have had one or two doses of vaccine and 4.1 million have had none.

As in previous years, the groups best protected are children 5 to 10 years of age, 84% of whom have had three or more shots of vaccine, and children 10 to 20 years of age, almost 80% of whom are now fully vaccinated.

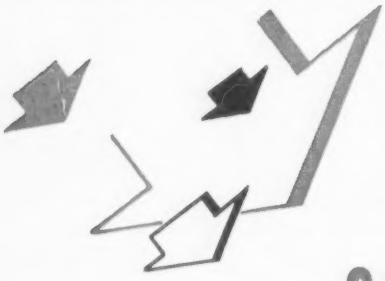
About 45% of persons 20 to 40 years old are now fully vaccinated, whereas, in 1959, only 36% had had three or more injections. Among persons in the 40 to 60 year age group, 83% have had no vaccine as compared to 86% in 1959.

HOPE Volunteers

American medical specialists from all parts of the country have volunteered to give up the comforts of the highest standard of living on earth to serve on the front lines of peace in Southeast Asia.

Dr. William B. Walsh, a Washington, D.C., internist and heart specialist, said that there is a waiting list of over 200 American medical personnel who have volunteered to serve on board the good ship HOPE, the American hospital ship currently in Surabaya, Indonesia.

In a luncheon speech at the National Press Club, where he received the United States Information Agency's Distinguished Service Citation, Dr. Walsh said that these medical specialists included doctors, dentists, nurses, pharmacists, physical therapists, and other medical personnel vitally



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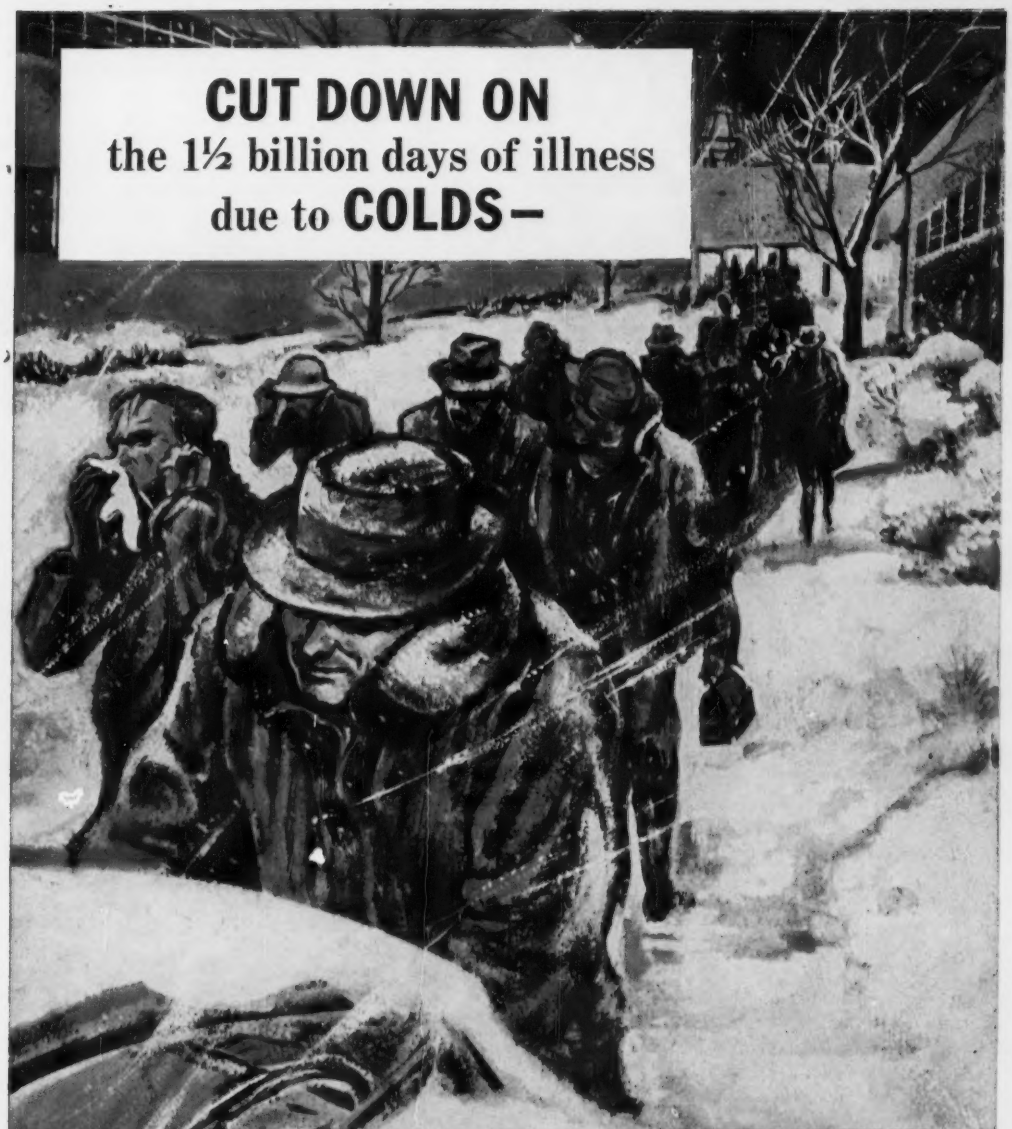

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needed by the newly developing countries.

Dr. Walsh said that this project has already been received with favor by the Indonesians, who invited the HOPE ship and her medical crew, and has scored a point for our side in the cold war.

"The HOPE has caused the East to react to the West for a change," Dr. Walsh said. "Premier Khrushchev, commenting on the flurry of favorable Indonesian editorial comment on the HOPE idea, found that he had to do something too, so he promised to build a hospital for the Indonesians."

Longevity Increased

America's older people have shared substantially in the marked reductions in mortality during recent decades, it is reported by statisticians of the Metropolitan Life Insurance Company.

Spectacular reductions in mortality among children and young adults have tended to obscure the progress at the older ages, the statisticians point out. However, a comparison of 1958's mortality rates at ages 60 and over with those of the 1929-31 period show marked reductions.

Among white men, a reduction was recorded at each age from 60 to 80 years, the decrease averaging about 13%. For example, at age 60 the death rate fell from 26.4 per 1,000 to 22.6 in 1958, and at age 75 the rate dropped from 85.3 to 73.3 per 1,000. Women have made considerably greater gains than men, the reductions ranging from as much as 47% at age 60, to more than 30% up through age 80.

Reductions in mortality have been accompanied by increases in longevity for older persons. In 1958 women at age 60 had an expectation of life of 19.2 years, a gain of 3.1 years since 1929-31. At age 80, the remaining lifetime averaged 6.4 years in 1958, a gain of 0.8 years. For men, the age 60 figure was 15.7 years in 1958, a gain of one year; and at age 80 life expectancy was 5.8, a gain of half a year.

A reflection of the improvement in mortality and longevity is seen in the increased chances of older people to survive an additional 10 years. For those currently reaching age 65, the chances of living at last another 10 years are almost 3 in 5 for men and 3 in 4 for women. Even at age 75, the chances of survival for 10 years are almost 1 in 3 for men and more than 2 in 5 for women.

"The improved record of longevity at the older ages reflects the remarkable advances in medicine and public health and their widespread application, the expansion of hospital facilities and services, better nutrition, fuller knowledge of personal hygiene, and the general rise in living standards," the Metropolitan statisticians conclude.

Try Yoga

Classes in Yoga are sponsored for employees at the Bird Machine Co., East Walpole, Mass. Yoga, an ancient form of psychotherapy, is supposed to relieve nervous tension and increase mental alertness.

—*Industrial Relations News*, Nov. 19, 1960.

Billion Dollar Hangover

A clinical psychologist has told management executives that they can help cure a "billion dollar hangover"—alcoholism in industry.

"The key contribution of industry is that it can provide a source of motivation for the alcoholic to seek help at a time when he is still working and therefore concerned with holding his job," said Harry Levinson of the Menninger Foundation.

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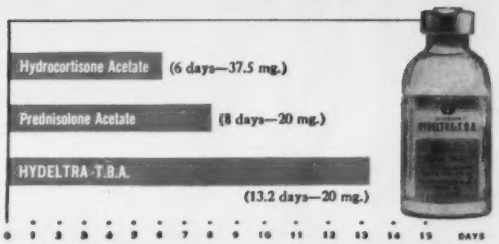
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"The company has a leverage that no one else can have. It can say to the alcoholic, while he is yet salvageable and still doing at least part of his job, 'Either you get help with your problem or else.'"

Mr. Levinson, Director of the Foundation's Division of Industrial Mental Health, addressed the fifth Alcoholism-in-Industry conference held at the La Salle Hotel. The conference was sponsored by the Chicago Committee on Alcoholism and the Chicago Association of Commerce. Referring to alcoholism as "slow suicide," he pointed out that it often seems as though the cards are stacked against the alcoholic.

This, he said, is because businesses and individuals too often bury their heads in the sand when the problem of alcoholism arises, because many psychiatrists and institutions will not treat alcoholics, and because some wives actually prefer for their husbands to be alcoholics.

In an earlier talk, Harrison Trice, Associate Professor in the New York State School of Industrial and Labor Relations of Cornell University, told the audience that it is harder to detect an alcoholic among management than among workers because the former tend to come to the office even when drunk or hung over while the latter stay home.

Alcoholics on managerial and professional levels come to work when suffering from their drinking out of a sense of duty, he said, to prove there is nothing wrong, and because they often are able to drink on the job. Their work performance, though, is mediocre on these occasions.

—*Chicago* (Ill.) *Tribune*, Dec. 4, 1960.

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1. Gordon, E. E. and Haas, A.: Industrial Medicine & Surgery 28:217, 1959.

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1. Crawford, O. B.: Anesthesiology 14:278, 1953. 2. Wiedling, S.: Xylocaine, The Pharmacological Basis For its Clinical Use, Stockholm, Almquist and Wiknell, 1959.

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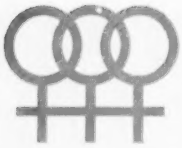

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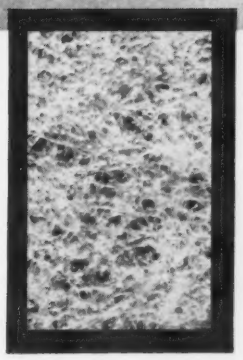

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